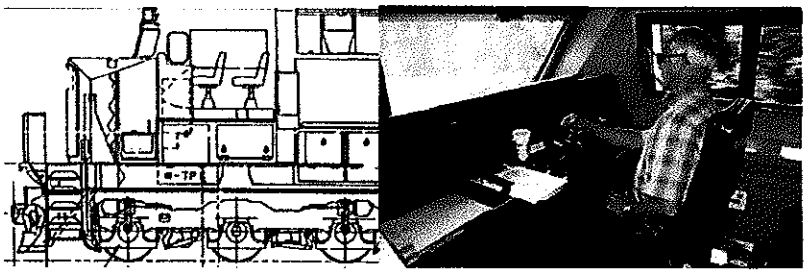

The results of the vibration and shock measurement of the basic x, y, z-axis ($a_{w\ rms}$) and vector sum (a_v) ranges were 0.07- 0.19, 0.13-0.4, 0.14-0.5 and 0.27 – 0.65, respectively. The ranges of the "shock" indicators MTVV/a_w and VDV/($a_w \cdot T^{1/4}$) were (x,y,z): 3.2-7.6, 2.9- 9.4, 3.3-10 and 1.44-2.3, 1.37-1.71, 1.44-1.94 and exceeded in a number of cases the critical values given by ISO 2631 (1). The daily equivalent static compression dose S_{ed} range was 0.11 to 0.79, mean 0.32 and the R-factor range was 0.12 to 0.92, mean 34, suggesting possible conflicting shock exposure risk information.

Discussion

Different shock indicator values were computed based on both ISO standards. Although, the new ISO 2631-5 method for evaluation of vibration containing multiple shocks suggests in our calculations possibly a low exposure risk other data and experience suggest an underestimation error relying solely on this indicator. We propose considering a combined sum score, in an overall risk assessment, that includes ergonomic co-factors such as awkward body posture, cab and seat design, and other environmental factors.

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CLINICAL ASSESSMENT AND CHARACTERISTICS OF MEN AND WOMEN EXPOSED TO HIGH LEVEL OF HAND-ARM VIBRATION

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Introduction

While the neurological and vascular aspects of Hand-Arm Vibration Syndrome (HAVS) has been generally accepted as a medical condition, the medical criteria and the clinical findings used to establish the diagnosis has been more difficult to bring to consensus. The criteria was first quantified by the Taylor-Palmear scale.[1] This criteria was subsequently modified in 1986 at the 1st Stockholm Workshop[2,3] to included more acceptance for the neurological effects that characterized the predominate findings in some workers. The relationship between hand-arm vibration and Carpal Tunnel Syndrome was defined in NIOSH 97-141[4].

While the aforementioned documents have defined the clinical entities associated with hand-arm vibration exposure, agreement on the clinical findings and test to confirm the diagnosis has be been more difficult to bring to consensus. Clinicians assessing HAVS has relied on a number of varied neurological and vascular tests. The neurological testing has focused on assessing damage to the sensory capability of the fingers for the neurological component including tests to measuring ability to sense vibration, cold or other end point finger sensor functions. However, the vascular testing has been traditionally focused on the ability to either measure vascular function or to reproduce the vascular blanching that occurs in HAVS with cold water provocation. Recent assessment of this testing in the United Kingdom Coal Miner's study has questioned the value of this testing especially in reviews by McGeoch.[5] In an attempt to provide some type of definitive testing to substantiate vascular damage from hand-arm vibration exposure, angiography is an alternative or adjunct to cold water provocation testing.

The standards that have been established to predict the level, type and incidence of HAVS have been based on clinical studies and reports that have essentially been all male populations. However, the recent entry of women into more vibration intensive jobs has brought about the exposure of some women to high levels of vibration previously only previously experienced by men. However, there have been only few studies that look at HAVS in women[6]. Although exposed the same vibration levels, it has not been clear that the latency and type of pathology of HAVS in women will be the same as for men.

The purpose of this study is to look at recent case studies of men and women exposed to jobs with high levels of hand-arm vibration with extensive clinical testing for both the neurological and vascular components of HAVS as well as other associated upper extremity conditions such as Carpal Tunnel Syndrome.

Methods

Clinical cases referred for evaluation with neurological testing including, vibrometry, Simmes-Weinstein mono filaments, 2 point discrimination, Purdue peg board testing and nerve conduction testing. Vascular testing included Allen's testing, Doppler studies of both upper extremities, cold water provocation testing and angiograph. Additional laboratory blood work and clinical examination was done to rule out alternative disease conditions that could confound results such as diabetes, collagen-vascular disease, etc.[6]

Results

Although the study was too small for statistical significance, review of the cases show that when exposed to the same high levels of hand arm vibration, women develop HAVS symptoms sooner than might be expected and early onset of Carpal Tunnel Syndrome.. In contrast men take longer to develop the same symptoms and are more likely to develop other finding such as tendonitis before they develop the constellation of symptoms and findings found in women.

Comparison of the vascular testing techniques indicates that the angiography can be helpful in confirming the vascular damage from hand-arm vibration exposure in both men and women. Furthermore, angiography may help localize areas of damage from specific exposure. The study proved to be too small to compare the effectiveness the various vascular testing techniques but suggest that further study is warranted.

Discussion

The study shows that there is a suggestion that present standards for the latency of HAVS and other vibration related disorders may be different for women then for men. Also review of clinical cases shows that angiography is useful tool in confirming and defining the level of vascular pathology in case of significant HAVS. Further enlarged studies to confirm both of these findings are recommended.

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ACUTE EFFECTS OF VIBRATION ON THE RAT-TAIL ARTERY

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Introduction

Acute vibration causes vasoconstriction in naïve human subjects[1]. Vibration-induced decrease in skin perfusion has also been reported in the rat-tail vibration model [2]. After vibration exposure, rat-tail arteries demonstrate vacuoles in smooth muscle cells, similar to that caused by pharmacological vasoconstrictors[3]. This study addressed the effects of different frequencies, durations and patterns of vibration on lumen size and vacuole formation using the rat-tail vibration model in male Sprague-Dawley rats (~300 g).

Methods

The different groups were: 4-hr continuous vibration at 30, 60, 120 and 800 Hz; continuous exposure durations of 5 min, 1 hr and 4 hr at 60 Hz; and 4-hr cumulative exposure of 60 Hz delivered intermittently in cycles of 10 min on and 5 min off. Acceleration was set at 49 m/s^2 r.m.s. for all frequencies. Unanesthetized rats were restrained in cages on a nonvibrating platform with their tails placed on a vibrating stage driven by a B&K motor (4809). The sham control animals were also placed in the vibration apparatus but not vibrated. Room temperature was controlled at 25 ± 1°C. Ventral arteries from proximal tail segments 7 were immersion fixed in aldehydes, embedded in epon-araldite and sectioned (0.5 µm) for morphological analysis. Vascular lumen sizes were measured as the percent ratio of the lumen perimeter to internal elastic membrane length using Image J software (NIH). The number of vacuoles in the smooth muscle layer of each artery section was counted.

Results

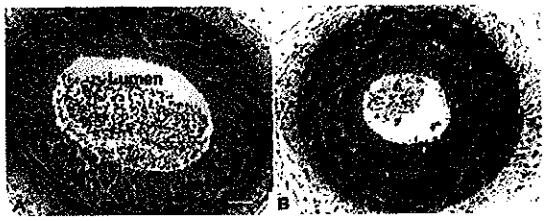
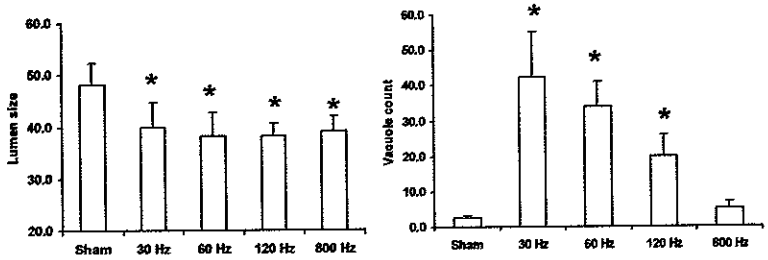


Fig 1: Semithin sections of arteries. A. Sham control. B. 4-hr vibration 60 Hz. In vibrated arteries, the lumen decreases in size, and smooth muscle cells (SMC) exhibit vacuoles (arrow). Bar equals 40 µm for each panel.

Fig 2: Bar graphs of lumen size and vacuole count when vibrated for 4 hrs at 30, 60, 120 and 800 Hz. * significantly different from sham, $p<0.05$.



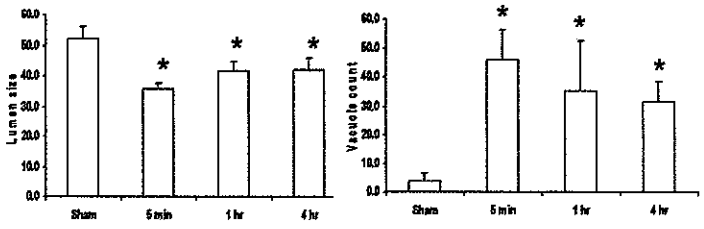


Fig 3: Bar graphs of lumen size and vacuole count when vibrated for 5 min, 1 hr and 4 hrs at 60 Hz. * significantly different from sham, $p<0.05$.

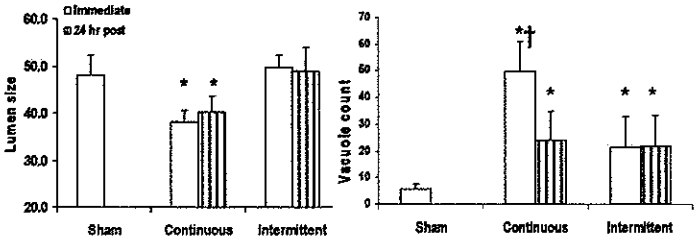


Fig 4: Bar graphs of lumen size and vacuole count when vibrated continuously or intermittently for 4 hrs at 60 Hz and examined immediately or 24 hr after exposure. * significantly different from sham, † significantly different from other vibrated groups, $p<0.05$.

Discussion

1. Vasoconstriction is induced by vibration at 30, 60, 120 and 800 Hz.
2. Vibration exposure of 60 Hz for 5 min is sufficient to cause vasoconstriction and generate smooth muscle cell vacuoles.
3. The decrease in lumen size persists at least 24 hrs after cessation of 60 Hz continuous vibration.
4. Both patterns of vibration, continuous and intermittent, cause the formation of smooth muscle cell vacuoles.

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EFFECTS OF REPEATED VIBRATION EXPOSURES IN MUSCLE TISSUE

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Introduction

Workers exposed to vibrating hand tools are at risk of developing symptoms such as cold-induced vasospasms, loss of tactile sensitivity, and loss of grip strength in the fingers and hands. These symptoms are known collectively as vibration white finger (VWF) or hand-arm vibration syndrome (HAVS). Symptoms of VWF or HAVS are in part due to repeated and prolonged peripheral vasoconstriction[1, 2]. The reduction in blood flow that occurs with vasoconstriction can result in oxygen deprivation (hypoxia) in soft tissues, such as nerves and muscle, and lead to functional and structural changes in these tissues. The present study examined muscle tissue to determine if vibration-induced changes in transcript levels and protein concentrations result in enhanced vasoconstriction and hypoxia. Manual dexterity was also assessed intermittently to determine if vibration-induced changes in cellular factors are accompanied by performance deficits.

Methods

An animal model was developed to study the biological and functional changes that occur in response to repeated segmental vibration exposures. In this model, the right paw of intact rats was exposed to a platform vibrated at a frequency 250 Hz and amplitude of 49 m/s^2 to simulate the vibration characteristics of hand-held grinders. Three groups of 8 rats each were studied: a vibration-exposed group, an exposure-control group, and a cage-control group. Exposure sessions, with or without vibration, were conducted 4 hr/day, 5 days/week for 5 weeks.

Manual dexterity was assessed intermittently during the 5-week exposure period with the Montoya stair-case test[3], which quantifies the rat's ability to reach for, grasp, and retrieve small food pellets placed below the rat on different levels or steps. Following the 5-week exposure period, the flexor muscles of the right forelimb were collected for analysis of gene expression, protein concentrations, and immunohistochemistry.

Results

Vibration-exposure resulted in an approximate 2-fold increase in the expression of α2C and α1D receptor transcripts in flexor muscles (Figure 1). These receptors mediate norepinephrine-induced vasoconstriction in smaller arteries. Vibration-exposure also resulted in an approximate 2-fold increase in hypoxia-induced factor-1α (HIF-1α), a transcription factor that is expressed in response to tissue hypoxia. Western analyses demonstrated that restraint caused a decrease in α1-receptor protein concentrations in the flexor, but vibration-exposure prevented the restraint-induced reduction (Figure 2). Immunohistochemistry performed on flexor muscles (not shown) demonstrated that α1 receptors are primarily located in arteries; maintained levels of these receptors could contribute to prolonged vasoconstriction following repeated vibration exposure. The staircase test showed some performance improvement, or a training effect, in manual dexterity for the control groups but not the vibration-exposed group (Figure 3).

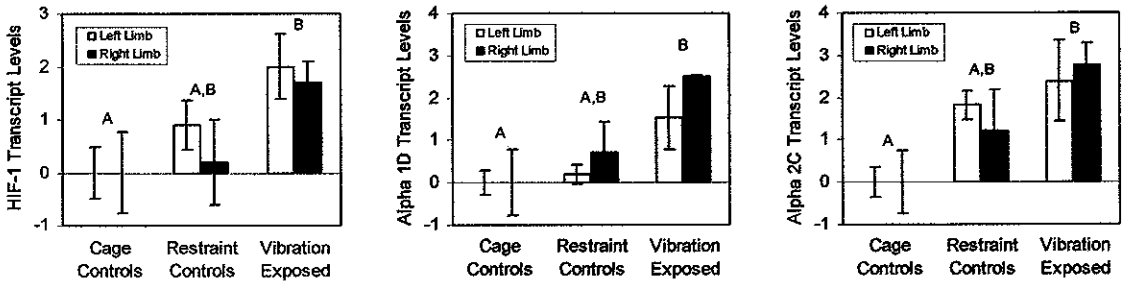

Figure 1. Relative gene transcript levels, expressed as mean fold change (±SE) in critical threshold from cage controls, for α2c, α1a/d, and HIF-1α in the left and right (exposed) limbs. Right limb is not significantly different from left; with right and left combined, A is significantly different from B.

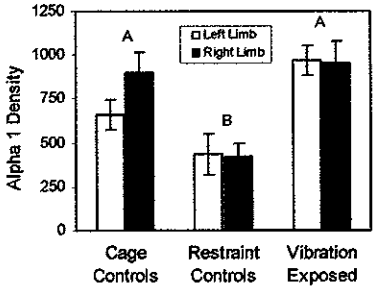

Figure 2. Mean relative optical density (±SE) of α1 proteins in the right flexor muscles as determined by western blot analysis.

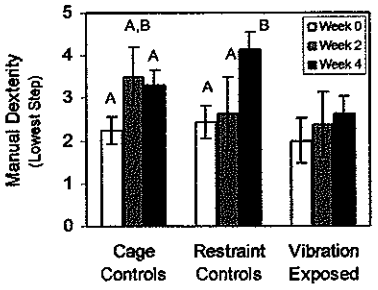

Figure 3. Staircase test of manual dexterity in the right limb (mean±SE; higher = better dexterity).

Results are consistent with the notion that vibration causes increased vasoconstriction in the vasculature, and subsequent damage or loss of function may be associated with hypoxia. Similar changes in transcript levels in both right and left limbs in the vibration-exposed group are consistent with reports of vibration-induced sympathetic vasoconstriction responses in contralateral (nonexposed) limbs[4]. Results also support the hypothesis that vibration-induced disturbances in motor control, manual dexterity, or loss of strength might be linked to hypoxia. A better understanding of these mechanisms can lead to the identification of early indicators of injury and improved methods for diagnosis and treatment of VWF or HAVS.

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VIBRATION EXPOSURE REDUCES NITRIC OXIDE CONCENTRATIONS IN THE VENTRAL ARTERY OF THE RAT TAIL

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Introduction

Vibration transmitted to the upper limb by the chronic use of hand tools can result in cold-induced vasospasms finger blanching and cyanosis, similar to that seen with Raynaud's phenomenon (4). These vasospasms, commonly referred to as vibration white finger (VWF), are in part the result of an increased sensitivity of peripheral arteries to the vasoconstricting effects of norepinephrine (e.g., (1-3)). However, alterations in vasodilating factors could also contribute to vasospasms. The goal of these studies was to determine if exposure to a single bout of vibration alters concentrations of the vasodilator, nitric oxide (NO), in a rat tail model of vibration. To determine if vibration exposure alters NO, we exposed animals to a single bout of vibration and measured concentrations of the synthetic enzymes, nitric oxide synthetase (NOS)-1 and NOS-3 in the ventral tail artery. We also directly assessed arterial concentrations of NO using a nitrate/nitrite assay.

Methods

General apparatus. Animals were placed in Broome-style restrainers, and their tail was secured to a vibrating or stable platform. Rats were exposed to a single 4 h bout of tail vibration (125 Hz, acceleration of 49 m/sec^2 r.m.s.) or restraint control. Animals were euthanized with an overdose of pentobarbital (100 mg/kg) and the ventral tail artery was dissected and frozen.

Experiment 1: Male Sprague Dawley rats (6 weeks old, n = 32) were used for all exposures. All animals were maintained in AAALAC accredited facilities, and all procedures were approved by the NIOSH Animal Care and Use Committee, and were in compliance with the CDC Regulations for the Care and Use of Laboratory Animals. Animals were euthanized 1 or 24 h after the completion of the exposure. Western analyses were performed on total proteins (80 µg/lane) isolated from the C16-18 artery segments. Band densities were detected by chemiluminescence and quantified using Scion Image, and analyzed using 2-way ANOVAs.

Experiment 2. Nitrate/nitrite concentrations.. Male Sprague Dawley rats (n = 24, 6 weeks of age) were maintained and exposed as described above. All animals were euthanized 24 h after the exposure and the ventral artery was collected. Nitrate/nitrite concentrations were measured in ventral artery tissue homogenates using the nitrate/nitrite colormetric Assay Kit (Caymen).

Results

Analyses of band densities revealed that there was an effect of time ($F(1, 17) = 6.03$, $p < 0.03$) on NOS-1 protein in arteries exposed to vibration, with NOS levels being lower in arteries collected 24 h after the exposure than arteries collected 1 h after the exposure. Although NOS-1 proteins concentrations were slightly lower in control arteries collected 24 h after an exposure than arteries collected 1 h after exposure, post-hoc contrasts indicated they were not significantly different than 1 h controls. In contrast, NOS-1 band densities from arteries collected 24 h after the exposure were lower than those collected 1 h after the exposure ($p < 0.01$; Figure 1). NOS-3

ACUTE VIBRATION INDUCES OXIDATIVE STRESS AND CHANGES IN TRANSCRIPTION IN SOFT TISSUE OF RAT TAILS

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Introduction

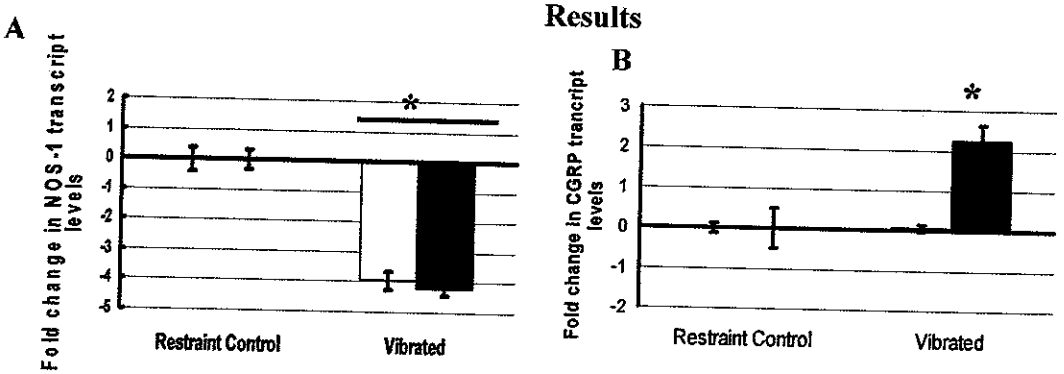
Repeated exposure to hand-arm vibration through the use of vibrating hand tools can result in the development of the disorder known as hand-arm vibration syndrome (HAVS; (1,3)). One of the hallmark symptoms of HAVS is cold-induced peripheral vasospasms that result in finger blanching (4). Although the vascular and neural pathology associated with vasospasms has been described, little is known about cellular mechanisms leading to this damage (4). To understand how vibration may alter vascular and neural physiology and anatomy, rats were exposed to a single bout of tail vibration and the molecular responses of neural and vascular tissues were measured to determine if there are immediate or sustained affects of vibration that may underlie longer term changes in physiology.

Methods

Experiment 1. Male Sprague Dawley rats (n = 32, 6 weeks of age) were housed in AAALAC accredited facilities. All procedures were approved by the NIOSH Animal Care and Use Committee and were in compliance with the CDC guidelines for care and use of laboratory animals. Vibration exposures were performed by restraining rats in Broome-style restrainers, and securing their tails to a vibration platform using 6 mm wide straps that were placed over the tail every 3 cm. Restraint control rats were treated in the same manner, except that the tail platform was mounted on isolation blocks and not on a shaker. The vibration exposure was 125 Hz, 49 m/s^2, for 4 h. Rats were euthanized with an overdose of pentobarbital (100 mg/kg) 1 h or 24 hours after the exposure. RTqPCR was used to measure transcript levels for endothelin 1 (ET-1), the 3 forms of nitric oxide synthetase (NOS) NOS-1, NOS-2, NOS-3 and norepinephrine receptor subtypes 1D, 2A, 2C, in artery tissue, and to measure calcitonin gene-related peptide (CGRP) and nitric oxide synthase-1 (NOS-1) in ventral nerves. Data were analyzed using 2-way ANOVAs.

Experiment 2. Male Sprague Dawley rats (n =24, 6 weeks of age) were maintained as described above. Animals were exposed to a single bout of restraint or vibration. Another group of animals served as cage controls. All animals were euthanized 24 h after the exposure, and tail arteries were isolated and frozen. Reactive oxygen species ROS) were measured using electron spin resonance spectroscopy (ESR). Arteries were homogenized over ice in 1 ml of PBS with protease inhibitor cocktail, using a tissue tearer (Biospecs Products Inc. Racine, WI USA). The sample was split into two 0.5 ml samples. One set had PBS and the spin label hydroxyl-TEMPO [0.1 mM] added while the other set had hydroxyl-TEMPO [1.0 mM] plus the specific hydroxyl radical scavenger, dimethylthiourea (DMTU), added to confirm the presence of the hydroxyl radical. Samples were vortexed and then placed in a flat cell for ESR analysis. The ESR spectrometer settings were: receiver gain, 6.32×10^2; time constant, 0.02 s; modulation amplitude, 1.0 G; scan time, 20 sec; magnetic field, 3490 ± 100 G (2). Data were analyzed using 1-way ANOVAs.

Results



Figures 1A-B. Fold changes in NOS-1(A) and CGRP (B) in the ventral tail arteries rats exposed to a single bout of vibration or restraint. The data are expressed as fold changes in transcript levels (mean ± sem) from the time matched controls. White bars represent transcript levels from tissue collected 1 h after the exposure and black bar represent transcript levels from tissue collected 24 h after the exposure (* different from time matched restraint control, $p < 0.05$). Exposure to vibration resulted in a reduction in NOS-1 transcript levels (main effect of exposure $F(1, 26) = 6.67$, $P < 0.02$) and an increase in CGRP transcript levels in nerve tissue collected 24 h after the exposure ($p < 0.05$).

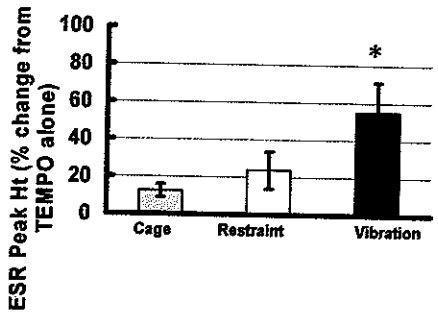


Figure 2. ROS measured using ESR. Acute vibration exposure resulted in an increase in hydroxyl radicals. The data to the left represent the ESR peak height when both TEMPO and DMTU were added to homogenates from the tail artery. The data are presented as the percent increase in peak height between TEMPO + DMTU and TEMPO alone. (mean ± sem; $p < 0.05$, different from cage and restraint controls; $F(1, 20) = 8.68$, $p < 0.002$).

Discussion

- Nitric oxide (NO) is a potent vasodilator produced by nerves and arteries. Vibration-induced reductions in the neural form of NOS, NOS-1, may result in reductions in NO synthesis and contribute to a prolonged noradrenergic-induced vasoconstriction.
- Increases in oxidative stress can result in a reduction in NOS activity. Acute exposure to vibration increase ROS in the arteries. This increase in ROS in arteries (and potentially nerves) may result in a reduction in NOS activity and NO production.
- The increase in CGRP transcript levels, which are not seen until 24 h after the exposure, may act to relieve the vibration induced vasoconstriction.

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VISUALIZATION OF MULTI-DIGIT MANIPULATION MECHANICS

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Introduction

Manipulation of hand-held objects in 3D space is a complex task. Understanding how individual digits interact with a hand-held object provides helpful information for hand tool designers, researchers, clinicians, and occupational therapists. At the object-digit interface, the contact mechanics can be represented by three force and three torque components. Six-component force/torque transducers can register all the three forces and three torques at the digit-object interface, and therefore are advantageous in the study of manipulation mechanics. The large number of force and torque signals from multiple force/torque transducers are difficult to interpret and therefore making experimental research of manipulation a challenging task. The purpose of this study was to develop a 3D visualization tool for the investigation of the contact mechanics at the object-digit interfaces during manipulation tasks.

Methods

A 3D stick-figure hand model was created based on digitized 23 anatomical landmarks of the hand. Five miniature 6-component force/torque transducers (4 × Nano17 for the fingers, 1 × Mini40 for the thumb, ATI Industrial Automation, NC) were used to record force and torque data at the tips of individual digits. Thirty channels of force/torque signals from the transducers were collected by a 16-bit analogue-digital converter (PCI-6031, National Instrument, Austin, TX) installed in a computer. The transducers were mounted on a custom-made rectangular aluminum handle for object manipulation. Coordinate frames were established at each transducer, on the handle, and at the base of the MicroScribe digitizer. To visualize the force vectors at the digit-tips, the coordinates of the hand landmarks in the MicroScribe coordinate frame and the force vectors in local transducer coordinate frames were transformed to a common coordinate frame defined on the handle. One healthy right-handed, male subject participated in the experimental study. During the tests, the participant sat in a chair by a testing table. The forearm was strapped to an arm holder in neutral rotation position. The instrumented handle was fixed on the testing table by a C-clamp through an adapting plate. With the hand of the subject gripped on the instrumental handle, the landmarks of the instrumented handle and the transducers, as well as the anatomical landmarks of the hand were digitized using the MicroScribe digitizer for the purpose of coordinate frame establishment and transformation as described above. The subject performed three different maximum isometric voluntary contraction tasks: (1) grasping, (2) rotating in pronation, and (3) lifting.

Results

The 3D hand model and representative force vector clusters in a single trial of grasping, rotating, and lifting tasks are shown in Figure 1. Each cluster was formed by displaying all the 3D force vectors during the period of "stabilized" maximum effort in a trial. The magnitude and

orientation of the force vectors of individual digits were strongly dependent on the task. Compared to the grasping and lifting tasks where forces were more evenly distributed among 4 fingers, forces in the rotating tasks were more concentrated on the two radial fingers, which was an advantageous strategy to produce pronation torque. In the grasping tasks, there was a trend that the force vectors of the four fingers converged. During the rotating tasks, there was a trend that the lower was the finger, the greater the projection angle, and the force vectors of the thumb pointed towards the ulnar aspect (-24.7 degrees). Therefore, the force vectors of individual digits tended to form a force couple to generate pronation torque. During the lifting tasks, the force vectors of all digits pointed upwards to generate maximal resultant uplifting force.

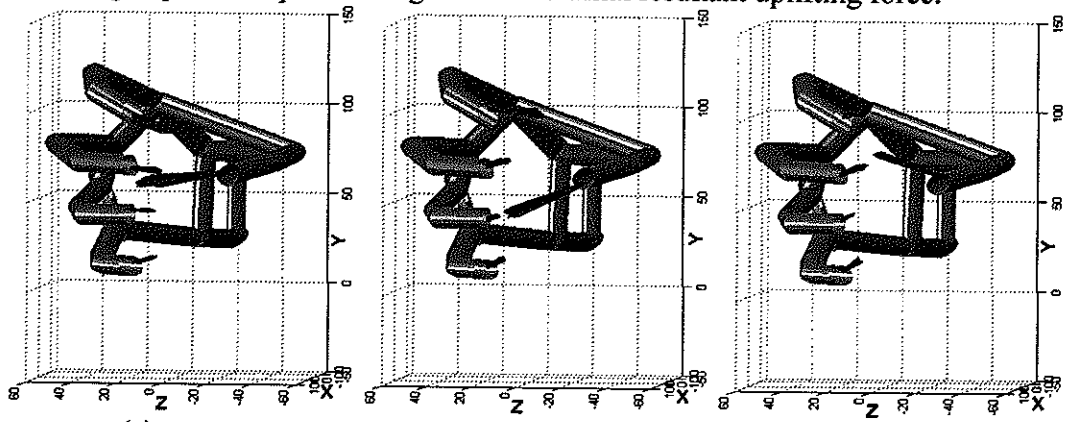

Figure 1. Three-dimensional hand model and representative force vector clusters at the digit-tips in tasks of (a) grasping, (b) rotating in pronation, and (c) lifting. Note that the magnitudes of all the force clusters within a task were equally scaled to achieve a reasonable visualization effects.

Discussion

The employment of 6-component force/torque transducers enabled us to construct 3D force vectors at the digit-object interfaces. Our preliminary results showed that during 5-digit manipulation, the human subject tended to maximize task efficiency by utilizing different force coordination strategies for different tasks. Complex force vector coordination patterns during manipulation tasks could be directly perceived through visualization. The 3D visualization tool developed in the current study could provide expedient and intuitive understanding of the mechanical interaction at the object-digit interfaces and the coordination among multiple digits. It could potentially be an effective tool for the understanding of human hand control and ergonomic designs that involve the usage of multiple digits. Further development of the current visualization tool will focus on incorporating kinematic data synchronized with force/torque measurement so that a relationship of the dynamic hand motion and manipulation mechanics could be established. The integration of force/torque data and kinematic data not only provides a dynamic visualization of grasping mechanics, but also allows for more advanced biomechanical studies such as the calculation of joint torques and muscle/tendon forces.

USE OF TUNGSTEN TO REDUCE VIBRATION EXPOSURE IN AIRCRAFT MANUFACTURING

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Introduction

Riveting operations in aircraft manufacturing involves the use of power tools for manually drilling holes for the rivets, power drills for the setting of the holes for the rivets, as well as rivet guns to drive and set the rivets. To close the rivet, the rivet is driven against a metallic bar commonly called a "bucking bar". The bucking bars are typically held firmly to increase the quality of the riveting, as well as keep the bucking bar from "dancing" against the metal piece being riveted. Thus, employees in aircraft manufacturing involved in riveting are exposed to hand-arm vibration from several sources, and epidemiological evidence suggests that vibration-related musculoskeletal disorders are associated with long term exposure to riveting tasks in the aircraft manufacturing of aircraft.[1,2] Recently, tungsten technology has been introduced into aircraft manufacturing for bucking bars, which are heavier than traditional steel bucking bars of the same size. Rivet guns with tungsten pistons instead of steel pistons have also recently been introduced with the objective of reducing vibration exposure to the riveter. The objective of this study was to assess vibration characteristics of steel and tungsten bucking bars and rivet guns to identify the combination that simultaneously reduced the combined exposure to both the "riveter" and "bucker".

Methods

Vibration (10g tri-axial accelerometer, Biometrics S2-10G-MF Series 2) was measured from eight experienced employees using seven different rivet guns on size 6 rivets, with the same person bucking for all subjects. Vibration was also measured on two different bucking bars for these same eight subjects, with the same person driving the rivets using the various rivet guns. The rivet guns consisted of three E4 steel piston guns with different RPMs (Guns A-E4, B-E4, C-E4), an E4 vibration dampened rivet gun (Gun D-E4D), an E3 steel piston rivet gun (Gun E-E3) and an E3 and E4 tungsten piston rivet guns (Guns F-E3T and G-E4T). The bucking bars were made of 90% tungsten (1694g) and cold-rolled steel (843g), and were the same shape and size. A two-way repeated measures analysis of variance was performed on the vibration (mean frequency weighted resultant acceleration) on both the rivet gun side and the bucking bar side, and mean rankings were used to assess the vibration simultaneously for the rivet gun and bucking bars to investigate which combinations provided the lowest vibration exposure.

Results

Frequency weighted resultant acceleration was significantly lower on the E3 tungsten (F-E3T) rivet gun than the E4 steel piston (B-E4) and the E4 tungsten piston (G-E4T) rivet guns (Figure 1). When measuring vibration on the bucking bar, the E4 (A-E4) steel piston rivet gun resulted in lower vibration on the bucking bars than the E4 tungsten piston (G-E4T) and E4 vibration dampened (D-E4D) rivet guns (Figure 2). Additionally, use of tungsten bucking bars resulted in a 35% decrease in resultant frequency weighted acceleration than when using steel bucking bars.

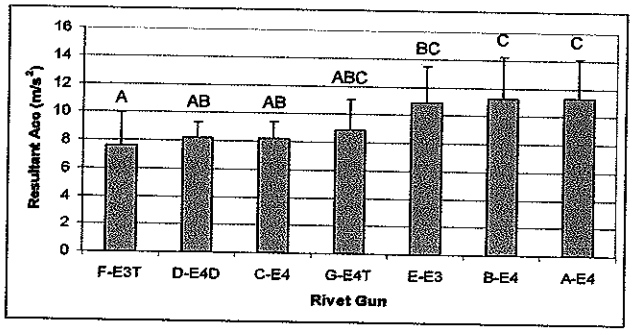

Figure 1. Resultant vibration measured on the rivet gun

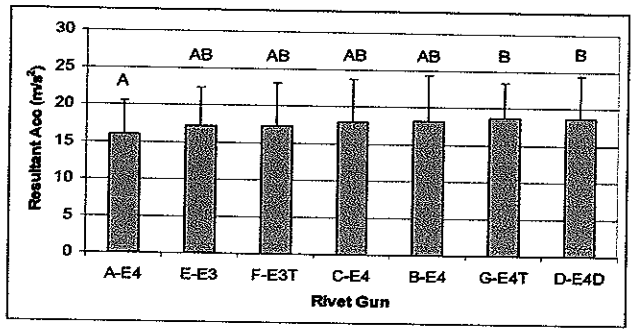

Figure 2. Resultant vibration measured on the bucking bar as a function of rivet gun used.

Discussion

Differences in vibration magnitudes were observed, however, the differences depended on whether the vibration was measured from the rivet gun or on the bucking bar. The vibration measured on the rivet guns indicated that the E3 (F-E3T) and E4 (G-E4T) tungsten piston rivet guns resulted in lower magnitudes, whereas E4 steel piston guns (B-E4 and A-E4) had higher magnitudes. Using tungsten bucking bars substantially decreased the vibration to the "buckers" compared to using steel bucking bars. However, the rivet guns that produced the lowest vibration to the riveter (dampened: D-E4D; tungsten: G-E4T) resulted in the highest vibration experienced on the bucking bar (Figure 3). Using the rankings on vibration levels for the tungsten bucking bar and different rivet guns to assess vibration exposure to the "riveters" and "buckers" simultaneously, using the E3 tungsten piston rivet gun (F-E3T) appears to reduce the vibration levels when considering both the riveting side and bucking bar side simultaneously when driving size 6 rivets. In conclusion, use of tungsten technology has the potential to reduce vibration exposure to riveters and buckers in certain riveting tasks.

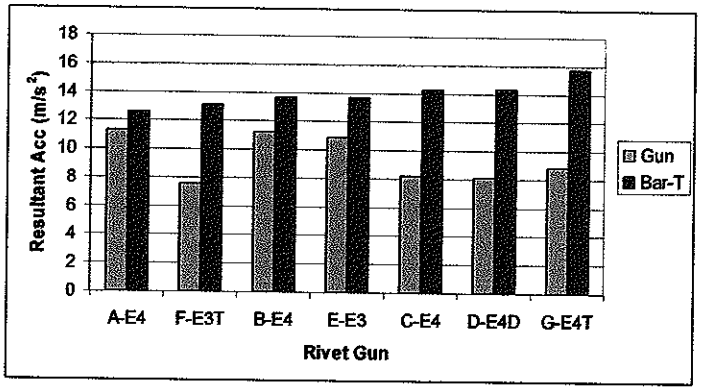

Figure 3. Resultant vibration measured on the rivet gun and the tungsten bucking bar.

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HANDLE DESIGN FOR OPTIMAL HAND FUNCTION

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Tubular Handles can negatively affect the contents of the carpal tunnel. Years of injuries from grasping handles for tools and machines can cause carpal tunnel syndrome, tendonitis and wrist joint injuries. The can cause inability to use a hand and resulted in the longer absences from work than injuries from falls, accidents or fires.[1]

Cylindrical, tubular and rectangular handles are rolled flat structures. They place the hand on a rolled flat surface where the ends of the middle and ring fingers overlap the index and small fingers. They are pulled along a series of lines that contact the end joint of the index finger, the middle bones of the middle and ring fingers and the end bone of the small finger. Cylinders are pulled diagonally in the hand toward the carpal tunnel (CT) area. Gripping in this manner tenses asymmetric muscle groups in the forearm.

Handles could work better if they do not place pressure on the CT and conform to the natural function or neutral hand position where the hand rests or dangles at the side of the body, the finger tips form a diagonal, the palm and fingers form a cup, the thumb rests between the index and middle fingers and the wrist is mildly extended. However, handles designed for the neutral position are pulled by diagonally oriented fingers into the valley between the thenar and hypothenar muscles where they can compress the median nerve and tendons exiting the CT.

Seven principles for handles that do not place pressure on the carpal tunnel and employ optimal hand position are presented. First, handles should align the ends of the fingers parallel to the horizontal crease and not diagonally. Second, handles should extend from the cupped fingers to meet the muscles at the base of the thumb on the radial side of the hand and extend further on the other or ulnar side to meet a portion of the small muscles. Third, handles should have a recess on the proximal side to prevent contacting or placing pressure on the carpal tunnel. Fourth, handle design should be based on hand measurements in a position of function. Fifth, handles should come in sizes. Sixth, handles should be placed on tools to maintain the wrist and elbow in neutral position. The seventh is handles should support the maximum area of the hand and absorb, but not direct, vibration to the carpal tunnel. These principles led to prototypes and patents for handles for gripping, pinching and squeezing[2,3,4].

The poster will illustrate and explain the principles Bonsil handles. Research, with existing tools, is needed to substantiate claims made for the Bonsil handle.

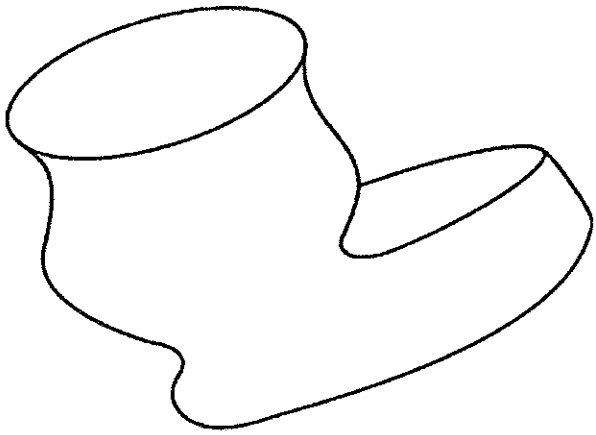

Figure 1

Figure 1 illustrates a large Bonsil handle. The upper section of handles for hammers will have a smaller radius than large power tools. Handles that support the upper body, such as crutches, canes and bicycles will have longer front to back lengths and shorter side to side lengths. The ulnar section extends further for supportive handles than for handles gripped like hammers.

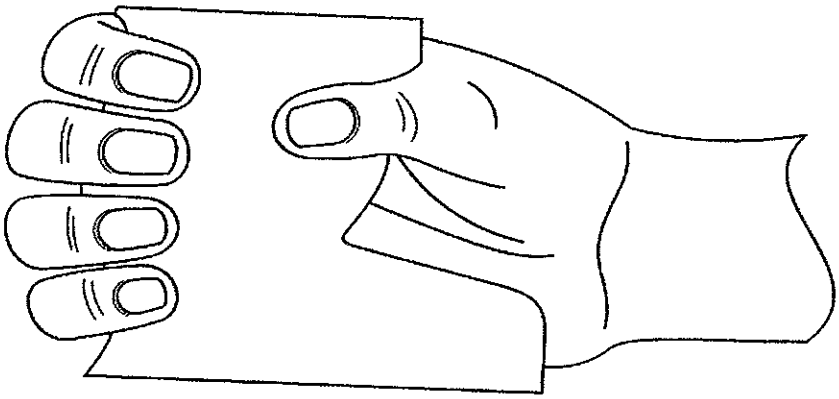

Figure 2

Figure 2 illustrates a hand wrapped around the Bonsil handle. Note, aligning the fingers preserves the cups formed by the fingers and palm. The thumb opposes the space between the index and middle fingers for strongest potential grip. The ulnar extension balances radial and ulnar grip. The CT area is not touched.

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3. US Patent 6,944,914 HANDLE AND FORCEPS/TWEEZERS AND METHOD AND APPARATUS FOR DESIGNING THE LIKE
4. US Patent 7,010,835 PARALLEL HANDLE SYSTEM AND METHOD FOR DESIGNING A PARALLEL HANDLE SYSTEM

VIBRATION TIME AND REST TIME DURING SINUSOIDAL VIBRATION EXPERIMENTS: DO THESE FACTORS AFFECT COMFORT RATINGS?

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Introduction

Industrial exposure to whole-body vibration is associated with injury and discomfort. Certain industries, notably mining, construction, and forestry, involve complex 6 degrees of freedom vibration. Laboratory-based studies of vibration are essential for controlled and systematic evaluation of the human responses to vibration[2]. The purpose of this pilot study was to evaluate whether the duration of the vibration exposure, and rest between vibrations, significantly influence the subjective ratings of comfort during laboratory-based studies of vibration.

Methods

Subjects: The cumulative vibration dose was calculated, and was below the health guidance caution zone recommended by International standards[3]. The experimental procedures were approved by the University of Guelph Research Ethics Board. Ten adult subjects participated in this pilot experiment. All subjects completed the entire experimental paradigm; no subjects complained of pain during or after the experiment.

Experimental Design: The experiment consisted of four blocks of vibration exposures; either 15 or 20 seconds of vibration (1 df:Z axis, 3 df:XY plane, 3df:YZ plane, or 6 df) alternating with either 5 or 10 seconds rest. The order of presentation of the four blocks was randomized. Each of the blocks was composed of 37 individual sinusoidal vibration exposures in randomized sequence. This abstract focused on ten identical trials, (6.3 Hz vertical vibration, 0.55 m/s^2 RMS) interspersed within each block, in order to assess whether the subjects' comfort ratings systematically varied between the 15 or 20 vibration exposures, the 5 or 10 second rest between vibrations, or within each block. The experiment involved 43 minutes of vibration within the 62 minute experiment.

Vibration Apparatus: A commercial parallel robotic platform was used to apply the specific vibration exposures (R2000, Parallel Robotics Systems Corporation, Hampton, New Hampshire). The subjects sat on a passenger seat from a 1992 Honda Accord that was rigidly mounted to the robotic platform (Figure 1). This robotic system performed the specific vibration exposures operating under closed-loop displacement control. A custom-written Matlab program automated the testing sequence.

Comfort Measures: Subjective feelings of comfort were verbally reported following each vibration exposure (during the rest period). The comfort scale was modelled after a previously published 9 point continuous comfort scale[1] which provided the greatest reliability and discrimination between different vibration intensities among 14 scales, but was modified to enable verbal reports (0 = "zero discomfort" & 8 = "max. discomfort").

Statistical Analysis: The raw comfort scale values for the ten identical vibration trials in each of the four blocks were analyzed using a three-way ANOVA.

Results

Figure 2 illustrates each of the subjects' comfort ratings for the ten repeated trials, collapsed across blocks of vibration duration. Statistical analysis did not observe significant interactions or main effects.

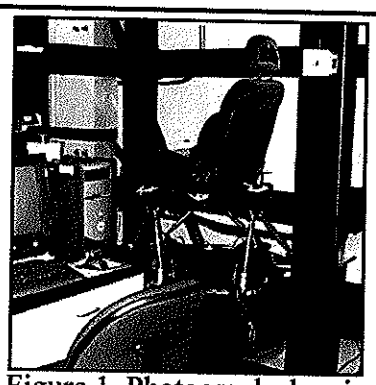

Figure 1. Photograph showing the seat mounted to the robot.

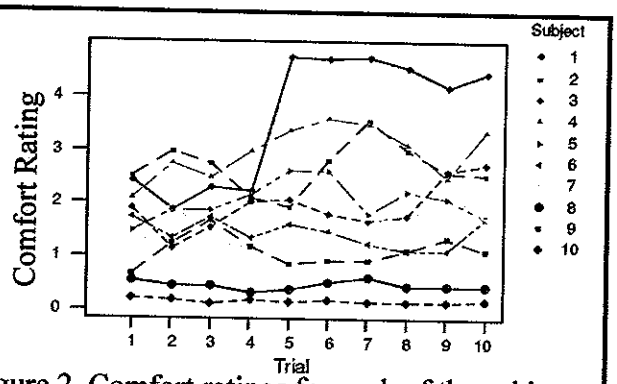

Figure 2. Comfort ratings for each of the subjects for the ten repeated trials.

Discussion

We did not observe statistically significant differences in comfort between the 15 or 20 second vibration exposures, or the 5 vs10 second rest durations. In addition, the comfort ratings did not vary systematically within the blocks of vibration. It appears that the one hour experiment duration did not result in systematic changes in reported comfort. This information is helpful for designing future laboratory-based vibration experiments.

Acknowledgements: Support provided by the Workplace Safety and Insurance Board of Ontario. The authors are grateful to the subjects for their participation.

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Industrial Health 2010, 48, 725–742

Original Article

Examination of the Frequency-weighting Curve for Accelerations Measured on the Seat and at the Surface Supporting the Feet during Horizontal Whole-body Vibrations in x- and y-Directions

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Abstract: In a laboratory experiment, six male subjects were exposed to sinusoidal (0.8, 1.6, 3.15, 6.3 and 12.5 Hz) or random octave band-width white noise (mid-frequencies identical to those of the sinusoidal vibrations) whole-body vibration in x- or y-directions, at six levels of magnitude (0.4, 0.8 and 1.6 m/s^2 r.m.s. non- and frequency-weighted) with two repetitions. In order to examine time effects, additional reference stimuli were used. Each subject was exposed to these 304 exposure conditions with a duration of about one minute on four different days (76 exposures per day). The subject's sensations of vibration intensity and vibration comfort were obtained by cross modality matching (length of a line). The subjects sat with an upright posture on a hard seat without backrest, hands on the thighs. The derived equivalent sensation contours suggest an underestimation of the sensation varying in extent from 2 dB to 8 dB at 1.6, 3.15, 6.3 and 12.5 Hz in comparison with the reference frequency 0.8 Hz for both types and directions of signals by the current evaluation methods according to ISO 2631-1 with the most pronounced effects revealed at the frequencies 3.15 and 6.3 Hz and at lower intensities (overall vibration total value a_w around 0.48 m/s^2 to 0.8 m/s^2 at the reference frequency 0.8 Hz).

Key words: Whole-body vibrations, Laboratory experiment, Frequency weighting, Subjective judgement

Introduction

The recently published report of the European Agency for Safety and Health at Work discovered a need for joint scientific efforts to clarify the prerequisite for an adequate risk assessment in the case of whole-body vibration (WBV). The implementation of the EC-directive 2002/44/EC[1)] intensified the discussion of the correctness of frequency-weighting curves and limit values for WBV. The evaluation methods concerning health risks, comfort and performance due to WBV, described in ISO 2631-1[2)] and used in application of the EU directive, are currently under critical discussion[3)].

ISO 2631 was first published in 1974 and later republished with new editorials and few corrections. An editorial combination of ISO 2631 (1978) and ISO 2631 AM 1 (1982b) resulted in ISO 2631-1 (1985). The version ISO 2631-1 (1997) replaced the earlier edition from 1985. The current frequency weightings in ISO 2631-1 (1997) were derived from meta-analyses of laboratory studies from the seventies of the last century. Frequency weightings obtained from equivalent discomfort contours are used for estimating the health risk as well, assuming an increase of risk with increasing vibration discomfort and pain, although this hypothesis has not been validated. However, the method is well established in practice. With an absence of information to the contrary, there seems to be no alternative method for health risk assessment.

Numerous experimental studies dealt with the effect of the frequency on discomfort caused by whole-body vibration[4–10)]. Inconsistencies in the obtained equivalent comfort contours might partially be explained by the dissimilar experimental methods (method of judging, sitting posture, seat, point of excitation etc.), but some divergences may have arisen from the different magnitudes of vibration that have been investigated.

Even in very early studies, significant major effects of acceleration and frequency and their interactions on discomfort or comfort ratings were obtained (Dempsey[11)], Oborne[12)]). These studies were limited to sinusoidal vertical vibration. Further investigations additionally included horizontal and roll, pitch and yaw vibrations. Parsons[13)] did not discover differences in levels described as "uncomfortable" between vibrations in the fore-and-aft and lateral axes. However, levels in these axes were found to be different from those obtained in the z-axis. In contrast to ISO 2631-1, mean discomfort caused by vertical acceleration showed only a small effect of frequency. Griffin[7)] concluded that the shapes of equivalent comfort contours need not normally depend on vibration level,

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possibly influenced by the choice of reference signal for magnitude estimation.

Griefahn and Bröde[6] used an intensity matching method. Applying the weighting of ISO 2631-1 they reported an underestimation of discomfort caused by sinusoidal horizontal WBV in y-direction in comparison with z-axis WBV for frequencies above 1.6 Hz. Maeda and Mansfield[8] reported a divergence of predicted and measured subjective ratings when ISO 2631-1 frequency weighting was used. Morioka[14] obtained significant interactions between vibration magnitude (0.02 to 1.25 m/s^2), frequency (2-315 Hz) and axis (x-, y- and z-axis) and concluded that probably no single linear frequency weighting can provide accurate predictions of discomfort caused by a wide range of magnitudes.

In different studies, various terms were used for judging the sensation caused by vibration. Griffin[15], Wyllie[16], Morioka[14] and Jang[17] asked the subjects to judge the "vibration discomfort". In ISO 2631-1, 1991, the phrase "effect of vibration on the comfort" is used. Jönsson[18] requested the subjects to rate the vibration on a scale from "uncomfortable" to "comfortable". In Europe, the use of these terms is linked with linguistic and semantic difficulties. For example, the word "discomfort" does not exist in German. The authors of the present study decided to ask for judgements of "vibration comfort" and additionally for the "vibration intensity" assuming that this phrase is less uncertain at least for German speaking subjects. Presumably, because of the same reasons, Griefahn[6] determined the "equal comfort contours" by asking the German speaking subjects to alter a vibration signal until they judged it to be equal in "magnitude" to a reference signal.

The theory of cross-modality matching used in the present study is based on investigations carried out by Stevens[19]. The authors found, that the association between the magnitude of the physical stimulus Φ and the sensation Ψ can be described by a power function.

(1) $\Psi = \Phi^m$

The power function can be logarithmised in order to get a linear association between lg Ψ and lg Φ:

(2) $\lg\Psi = m \times \lg\Phi$

The factor m is the so called "Stevens' exponent". Stevens determined these exponents for different types of stimuli. The subjects were asked to assign a number to a stimulus representing its sensation. This judging method is called "magnitude estimation". As a result of these experiments, the exponent can be assumed to be only dependent on the type of stimulus and nearly constant, provided the task is identical for all subjects and conditions when external influences on the judgements are absent or constant.

In contrast, cross-modality matching is based on the subjects' ability to judge their sensation according to the sensation caused by another stimulus. For example, the subjects could be requested to adjust the length of a line (response modality) according to a sensation caused by a simultaneous vibration (stimulus). This equilibrium of stimuli (exposed stimulus and scalable stimulus adjustable by the subject e.g. the brightness of an area, length of a line, force of a hand grip) can be influenced by other conditions (additional stimuli).

This procedure can be mathematically described as follows:

(3) $\Psi_1 = \Phi_1^{m1}$
power function of the stimulus which has to be judged (e.g. vibration)

(4) $\Psi_2 = \Phi_2^{m2}$
power function of the response modality (e.g. length of a line)

Provided that the sensation concerning stimulus and response modality are equalised with respect to the question which has to be answered (e.g. intensity or discomfort or annoyance):

(5) $\Psi_1 = \Psi_2$, therefore follows

(6) $\Phi_1^{m1} = \Phi_2^{m2}$

Logarithmised in order to get linear associations:

(7) $m_1 \times \lg \Phi_1 = m_2 \times \lg \Phi_2$ and finally

(8) $\lg \Phi_2 = m_1 / m_2 \times \lg \Phi_1$

In the present study, the vibration stimuli were judged by adjusting the length of a line presented on a screen simultaneously with the vibrations, in accordance with the sensations. The Stevens' exponent is m_2=1 for a length of a line[19]. Therefore, the determined exponents could be directly compared with those obtained by magnitude estimation in previous studies[9, 14, 20].

Exposure to whole-body vibration shall be assessed on the basis of frequency-weighted accelerations and multiplying factors in accordance with ISO 2631-1. For the evaluation of the effect of vibration on comfort, the weighted root mean square acceleration shall be determined for each axis of translational vibration at the surface which supports the person. For seated persons and horizontal seat surface vibration, the frequency weighting W_d should be applied with the multiplying factor k=1. The point vibration total value a_v shall than be calculated by a root-sum-of-squares summation. Alternatively, where the comfort is affected by vibrations at more than one point an overall vibration total value a_{ov} can be determined from the root-sum-of-squares of the point vibration total values. In this case, vibration at the feet is recommended to be assessed using the frequency weighting W_k and the multiplying factor k=0.25.

The study aimed to examine the effects of sinusoidal and random whole-body vibration in x- and y-axis on the perceived intensity and comfort. The equivalent intensity and comfort contours predicted on the basis of the overall vibration total value a_{ov} at different vibration magnitudes were compared to the current evaluation methods according to ISO 2631-1.

Subjects and Methods

Subjects and posture

In a laboratory experiment, six male subjects were exposed to whole-body vibrations of different magnitudes, frequencies and types of vibration signal. In order to determine the optimal sample size, information about the variance of the dependent variables is necessary. The authors have already performed similar investigations using cross-modality matching for the subjective judgements, but the vibration signals and seats were not comparable with those in the current study. However, the authors understood from their previous experi-

ence[21, 26] that six subjects should be sufficient for discovering significant differences in the mean values of intensity or comfort judgements due to different vibration magnitude levels. There were no available data obtained by the authors concerning the effects of different frequencies, directions and types of vibration signals, using the same method in previous studies. Data analyses of the present study discovered that the number of subjects was sufficient for observing the expected differences due to vibration magnitude and frequency, but the current paper does not focus on this topic. A publication on this issue is in preparation.

The subjects were selected from a then available 36-person subgroup of a larger dataset consisting of 100 subjects with fixed subject numbers (the numbers did not change after the selection). Therefore, a subject with number 37 appears in Fig. 1. In order to guarantee the subjects' suitability and to equalize the physical prerequisites, the results of medical and anthropometric examinations including a list of contraindications were used. The ages varied from 24 to 46 yr (mean value 31 yr), the heights from 177.7 cm to 188.5 cm (mean value 183.9 cm), the body masses from 72 kg to 94.3 kg (mean value 84.5 kg) and the body mass indices from 20.7 to 27.8 (mean value 25.0). The individual values are shown in Fig. 1. Previous studies indicated that a similar understanding of semantic nuances is favourable for the comparability of subjective judgements. Therefore, subjects with comparable educational level were chosen. Moreover, comprehensive experience in driving might influence the subjective judgements (Seidel *et al.*[29]). For that reason, professional drivers were excluded. It could be of interest to investigate differences in subgroups of different professions, but a study design of that kind would be very time-consuming and expensive.

The subjects sat with an upright posture on a hard seat without backrest, with hands on the thighs.

The Ethics Committee of the Berlin General Medical Council approved the experiments. Informed consent was obtained from all subjects.

Vibration exposure and measurements

The experiment was conducted with a six-degree-of-freedom (DOF) servo-hydraulic simulator with a control system by FCS Control Systems B.V. (The Netherlands) in the vibration laboratory of the Federal Institute for Occupational Safety and Health, Berlin, Germany, considering the guidelines for human experiments with WBV (ISO 13090-1, 1998). Drive files were generated and optimised to realize the desired accelerations. The translational accelerations were measured on the platform and on the seat in three axes (accelerometer Type Endevco 7290A-10) with a sampling frequency of 1 kHz.

The subjects were exposed to sinusoidal (five frequencies 0.8, 1.6, 3.15, 6.3 and 12.5 Hz) or random octave band-width white noise (mid-frequencies identical with those of sinusoidal vibration) whole-body vibration in x- or y-directions, at six levels of magnitude (0.41, 0.82 and 1.65 m/s^2 desired overall vibration total value non-weighted $a_{des,ov(n.w.)}$ (n.w. - M1, M2 and M3) and frequency weighted $a_{des,ov}$ (w. - M4, M5 and M6)) with two repetitions. Table 1 shows the desired – not the measured - accelerations in the main axes. Magnitudes M4, M5 and M6 with desired overall vibration total values $a_{des,ov}$ weighted according to ISO 2631-1, were chosen to be numerically equal to the modified non-weighted desired overall vibration total values $a_{des,ov(n.w.)}$ M1-M3 (grey lines in Table 1).

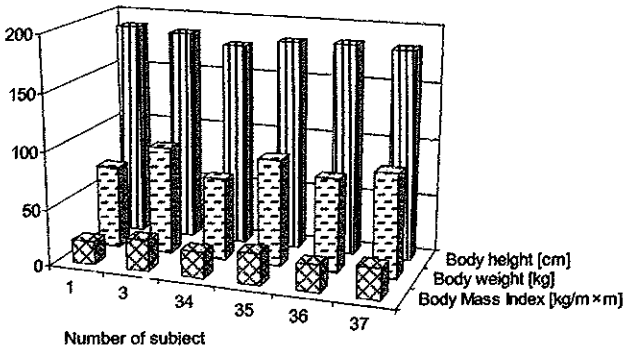

Fig. 1. Body Mass Index, weight and height of the six selected volunteers.

Table 1. Levels of the root mean square (r.m.s.) values of the desired non-weighted acceleration in the axes of excitation on the seat and at the feet $a_{des,ex,seat,\ feet}$ and calculated desired overall vibration total values $a_{des,ov}$ weighted according to ISO 2631-1 (Eq. (9)) and modified without frequency weighting $a_{des,ov(n.w.)}$ (Eq. (10)), sinusoidal (F) or random octave band-width white noise (B) in m/s^2 with magnitudes M).

	Frequency [Hz]	0.8	1.6	3.15	6.3	12.5
Magnitude [m/s^2]		F1/ B1	F2/ B2	F3/ B3	F4/ B4	F5/ B5
M1	$a_{des,ex,seat,\ feet}$	0.40	0.40	0.40	0.40	0.40
	$a_{des,ov(n.w.)}$	0.41	0.41	0.41	0.41	0.41
	$a_{des,ov}$	0.40	0.39	0.27	0.17	0.11
M2	$a_{des,ex,seat,\ feet}$	0.80	0.80	0.80	0.80	0.80
	$a_{des,ov(n.w.)}$	0.82	0.82	0.82	0.82	0.82
	$a_{des,ov}$	0.80	0.78	0.54	0.33	0.22
M3	$a_{des,ex,seat,\ feet}$	1.60	1.60	1.60	1.60	1.60
	$a_{des,ov(n.w.)}$	1.65	1.65	1.65	1.65	1.65
	$a_{des,ov}$	1.60	1.56	1.08	0.67	0.44
M4	$a_{des,ex,seat,\ feet}$	0.41	0.42	0.61	0.99	1.47
	$a_{des,ov(n.w.)}$	0.42	0.43	0.63	1.02	1.52
	$a_{des,ov}$	0.41	0.41	0.41	0.41	0.41
M5	$a_{des,ex,seat,\ feet}$	0.82	0.84	1.22	1.97	2.96
	$a_{des,ov(n.w.)}$	0.85	0.87	1.26	2.03	3.05
	$a_{des,ov}$	0.82	0.82	0.82	0.82	0.82
M6	$a_{des,ex,seat,\ feet}$	1.65	1.69	2.46	3.95	5.96
	$a_{des,ov(n.w.)}$	1.70	1.74	2.54	4.07	6.14
	$a_{des,ov}$	1.65	1.65	1.65	1.65	1.65

The idea behind this study design was to perform the investigation twice, with both non-weighted and weighted values. Assuming that the frequency-weighting curves recommended in ISO 2631-1 correctly reflect the sensations, the shape of the frequency-weighting curves derived from the sensations due to the non-weighted magnitude levels M1 to M3 should coincidence with the current weighting curves. The equivalent sensation contours and frequency-weighting curves derived from

Table 2. Experimental design

Day of experiment	Direction of exposure	Repetition	Reference stimuli
1	Y	1	M2F3X and Z
2	Y	2	M2B3X and Z
3	X	1	M2B3Y and Z
4	X	2	M2F3Y and Z

X, Y and Z=axes of excitation, M=magnitude, F=sinusoidal vibration, B=random octave band-width white noise.

Table 3. Exposure conditions used for subject 1 at experimental day number one

Trial 1	Trial 2	Trial 3	Trial 4
M2F3X	**M2F3X**	**M2F3X**	**M2F3X**
M2F3Z	**M2F3Z**	**M2F3Z**	**M2F3Z**
M6F1Y	M4F2Y	M4B5Y	M5B3Y
M6B1Y	M2F1Y	M2F3Y	M6B4Y
M5B1Y	M4B2Y	M4F5Y	M6F3Y
M1F2Y	M1B1Y	M2B3Y	M6F4Y
M5F1Y	M5F2Y	M3B5Y	M6B3Y
M1B2Y	M1F1Y	M3F3Y	M5B4Y
M4B1Y	M5B2Y	M3F5Y	M1F4Y
M2F2Y	M6B5Y	M3B3Y	M5F4Y
M4F1Y	M6F2Y	M2B5Y	M1B4Y
M2B2Y	M6F5Y	M4F3Y	M4B4Y
M3B1Y	M6B2Y	M2F5Y	M2F4Y
M3F2Y	M5B5Y	M4B3Y	M4F4Y
M3F1Y	M1F3Y	M1B5Y	M2B4Y
M3B2Y	M5F5Y	M5F3Y	M3B4Y
M2B1Y	M1B3Y	M1F5Y	M3F4Y
M2F3X	**M2F3X**	**M2F3X**	**M2F3X**
M2F3Z	**M2F3Z**	**M2F3Z**	**M2F3Z**

bold letters = reference stimuli.

the weighted magnitude levels M4 to M6 should be horizontal lines.

In Table 1, the desired overall vibration total values were calculated in accordance with equation (9) and equation (10), assuming the accelerations in the cross axes were zero.

To examine the time effects, 16 additional reference stimuli were used per day. Every subject was exposed to these 304 exposure conditions on four different days, 76 single exposures per day, randomized and divided into 4 trials of 19 single exposures (Table 2 and Table 3). Each single exposure had a duration of about one minute (Fig. 2). There were short pauses between the single exposures. Therefore, one trial lasted approximately 25–30 min. The subjects were asked to walk or stand during the 10 min pause between the trials. Altogether, it took roughly two hours to two and a half hours to realize the 76 single exposures per day. Day 2 and day 4 were complete repetitions of day 1 and day 3, respectively, but the exposure conditions were presented in a different order. No sequence of exposure conditions was used twice. The experimental design was described in more detail in Kreisel *et al*[28].

(9)

$$a_{des,ov(n.w.)} = \sqrt{a^2_{des,ex,seat} + 0.25^2 \times a^2_{des,ex,feet}}$$ modified

with

$a_{des,ex,seat}$ = desired non-weighted acceleration (r.m.s.) on the seat in the axis of excitation and

$a_{des,ex,feet}$ = desired non-weighted acceleration (r.m.s.) at the feet (platform) in the axis of excitation

(10)

$$a_{des,ov} = \sqrt{a^2_{des,Wd,ex,seat} + 0.25^2 \times a^2_{des,Wk,ex,feet}}$$

according to ISO 2631-1

with

$a_{des,Wd,ex,seat}$ = desired weighted acceleration (r.m.s.) on the seat in the axis of excitation and

$a_{des,Wk,ex,feet}$ = desired weighted acceleration (r.m.s.) at the feet (platform) in the axis of excitation

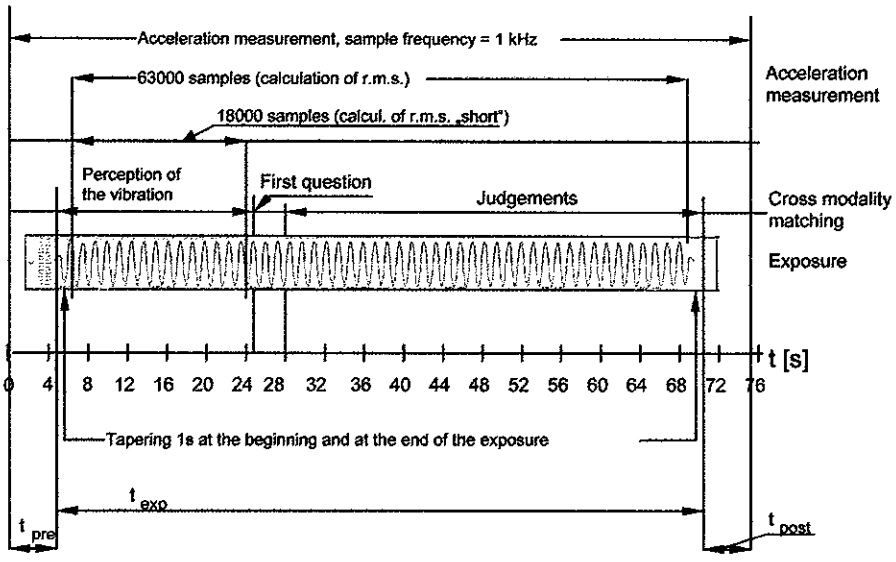

Fig. 2. Measurement of acceleration and subjective judgements during one single exposure.

and assumption: accelerations in the cross axes equal zero in both equations

The combination of the frequency-weighting curves W_d and W_k and the multiplying factors k=1 (seat) and k=0.25 (feet) is defined as $W_d \wedge W_k$ in this paper for convenience, using the Boolean operator for conjunction ($\wedge$). It is valid for the calculation of the desired overall vibration total value in this experiment. Supposing identical exposure on the seat and the platform due to the mechanical stiffness between seat and platform and assuming that the accelerations in the cross axes were zero, equation (11) was derived from equation (10).

(11)

$$a_{ov} = a_{ex} \times \sqrt{1.0^2 \times W_d^2 + 0.25^2 \times W_k^2} = a_{ex} \times (W_d \wedge W_k)$$

with

W_d and W_k = weighting factors according to ISO 2631-1, Table 3 and

a_{ex} = non-weighted acceleration (r.m.s.) on the seat and at the feet (platform) in the axis of excitation

The sensations of vibration intensity and vibration comfort were obtained by cross-modality matching (length of a line). The subjects responded by adjusting the length of a line presented on a screen in front of them. They were instructed to adjust the length of the line in accordance with their sensations, i.e., the stronger the sensation the longer the line had to be. The subjects used a mouse which was fixed on the vibration simulator to be easily gripped with their right hand (Fig. 3). The cross-modality matching included answers on the following questions:

How intensive do you perceive the vibration to be?

How comfortable do you perceive the vibration to be?

Day 1 started with a training session with at least 10 different representative exposures to allow the subjects to reach a similar level of experience. No subject expressed having been restricted due to the maximum length of the presented line (1,481 mm) on a screen at a distance of 2,600 mm from the subject's eyes, neither during the training session nor during the main study. At the beginning of each experimental day, the subjects had to read a written instruction (Appendix B). The insructions were repeated by the operator at certain time points during the trials.

Data analyses

Data were examined with the statistical program SPSS 15.0.1. The order of successive steps in the data analyses is illustrated in Fig. 4.

The overall vibration total values calculated from the measured accelerations $a_{ov(n.w.)}$ and a_{ov} (Eq. (12) and Eq. (13)) differed from the desired overall vibration total values $a_{des,ov(n.w.)}$ and $a_{des,ov}$ according to Table 1 in about 10% of the cases by 1 dB or more. Therefore, these excitations and the corresponding responses (length of lines) were treated as missing values when differences of mean values between the responses caused by different exposure levels or time points had to be examined (*t*-Tests and Variance Analyses). The excitations in the higher frequencies were most frequently concerned. The cross-axis vibration reached a maximum of 31.7% (y-axis during excitation in x-axis) and 26.6% (x-axis during excitation in y-axis) for sinusoidal excitation at 12.5 Hz, calculated on the basis of the mean values of the r.m.s. of the measured accelerations in main and cross axes. Information about the background vibration is given in Appendix A.

(12)

$$a_{ov(n.w.)} = (a_{x,seat}^2 + a_{y,seat}^2 + a_{z,seat}^2 + 0.25^2 \times a_{x,feet}^2 + 0.25^2 \times a_{y,feet}^2 + 0.4^2 \times a_{z,feet}^2)^{1/2}$$

modified

with

$a_{x,seat}$, $a_{y,seat}$, $a_{z,seat}$ = measured non-weighted acceleration (r.m.s.) on the seat in the x-, y- and z-axis and

$a_{x,feet}$, $a_{y,feet}$, $a_{z,feet}$ = measured non-weighted acceleration (r.m.s.) at the feet (platform) in the x-, y- and z-axis

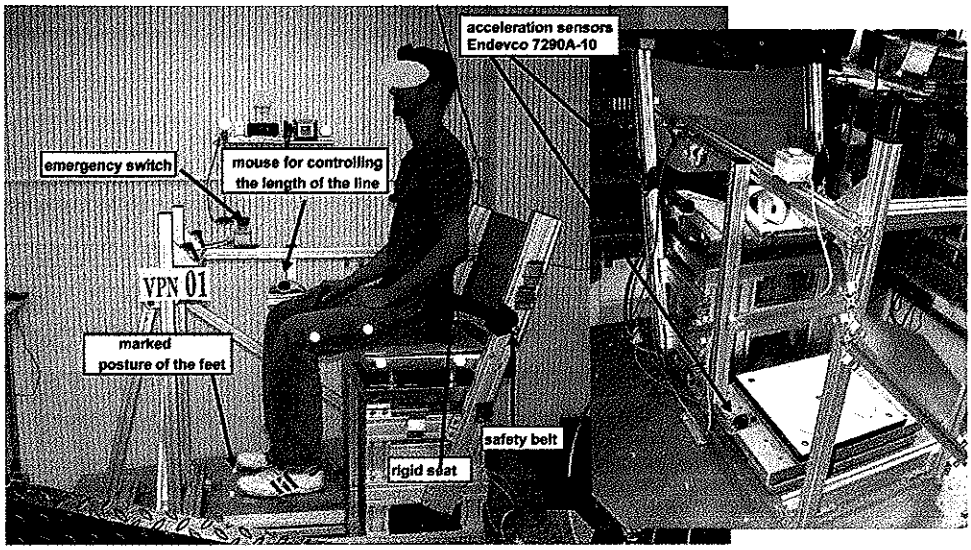

Fig. 3. Subject sitting on the rigid seat and location of the acceleration sensors.

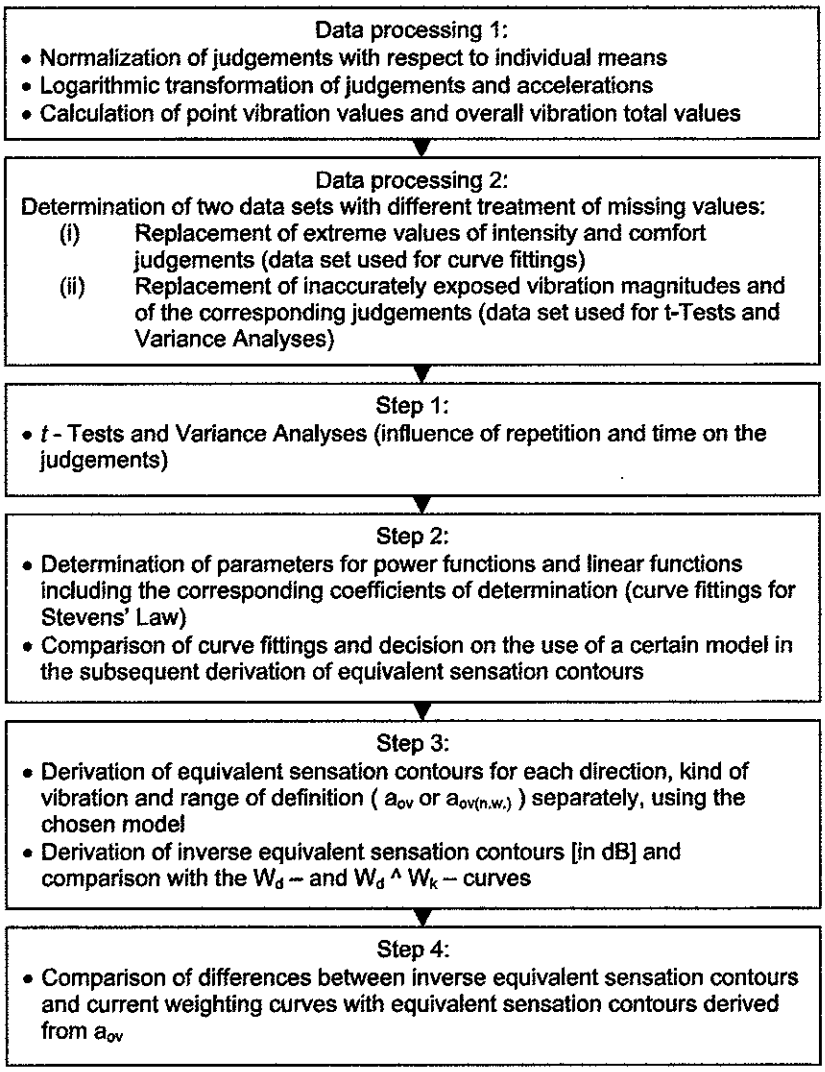

Fig. 4. Order of successive steps in data processing and analyses.

(13)

$$a_{ov} = (a^2_{Wd,x,seat} + a^2_{Wd,y,seat} + a^2_{Wk,z,seat} + 0.25^2 \times a^2_{Wk,x,feet} + 0.25^2 \times a^2_{Wk,y,feet} + 0.4^2 \times a^2_{Wk,z,feet})^{1/2}$$

according to ISO 2631-1

with

$a_{Wd,x,seat}$, $a_{Wd,y,seat}$, $a_{Wk,z,seat}$ = measured weighted acceleration (r.m.s.) on the seat in the x-, y- and z-axis and

$a_{Wk,x,feet}$, $a_{Wk,y,feet}$, $a_{Wk,z,feet}$ = measured weighted acceleration (r.m.s.) at the feet (platform) seat in the x-, y- and z-axis

For curve fittings, only the extreme values of the length of the lines were considered as missing values (intensity: 1 value, comfort: 16 values out of 1,440 single exposures in the main directions x and y without reference stimuli). In contrast to the variance analyses and t-tests, the differing excitations and the corresponding responses were not excluded from the linear regressions analyses.

Values normalized with respect to individual means per experimental day or individual means over all days were derived from the length of the lines measured as pixels. In addition, a logarithmic transformation of data was performed. Because of the simultaneous exposure to vibration on the seat and at the feet in the experiment and the instruction to judge integratively the entire vibration exposure, the judgements were assumed to be reflected more accurately by the overall vibration total value a_{ov} than by the point vibration total value a_v or the vibration in the axis of excitation only. Consequently, the relations between the a_{ov} and the subjective judgements were determined by curve fitting to power functions and linear associations for each frequency, direction and type of vibration signal separately in order to test the agreement with the Stevens' law.

It is a point of discussion, whether the modified overall vibration total value used in this study is valid (Eq. (9) and Eq. (12)). The multiplying factors k for multiple input locations were applied without frequency weighting of the input signals. As mentioned in the discussion later, there seems to be a lack of literature concerning an exact explana-

tion of methods which were used to derive the frequency-weighting curves and the multiplying factors k recommended in ISO 2631-1. An appropriate experiment should strictly differentiate between input location effects and frequency effects and a combination of both influences. Moreover, for estimation of the effect of vibration on the comfort according to the standard, the point vibration total value 'shall be calculated' and the overall vibration total value 'can be determined' (ISO 2631-1, paragraph 8.2.3). Griffin[15] mentioned that there is 'a conceptual problem in the choice of the frequency, axis and input position weightings when evaluating the vibration which occurs at several input positions' (Griffin[15], page 82). The effects of relative motions between body parts due to excitations in different directions and at different input points are complex and hardly predictable. At present, there is no standardised evaluation method which considers this fact. Further considerations seem to be necessary. In fundamental investigations, the body parts are often separately exposed in one direction. The curves of equivalent sensation are subsequently derived on the basis of the point vibration total value on the seat $a_{v,seat,(n.w.)}$ or even the non-weighted acceleration in the main axis only, neglecting the cross-axis vibration. Considering the multiplying factors is not necessary in these studies. In the current investigation, seat and feet were exposed simultaneously and identically. It would have been inaccurate if the effect of the exposure at the feet was not taken into account. The authors decided to suppose an influence of the vibration at the feet to be smaller than that on the seat regardless of whether the input signals were frequency-weighted or not. In the absence of further scientific findings the recommended multiplying factors k=0.25 (x- and y-axis) and k=0.4 (z-axis) were applied (Eq. (12) and (13)). However, for mathematical reasons, applying the non-weighted forms of (i) the overall vibration total value $a_{ov,(n.w.)}$ (Eq. (12)), or (ii) the point vibration total value on the seat $a_{v,seat,(n.w.)}$ or (iii) the root-sum-square of $a_{v,seat,(n.w.)}$ and $a_{v,feet,(n.w.)}$ without using the multiplying factors to the linear regressions (see Eq. (14) and Eq. (16)) do lead to identical results. The difference between the levels of magnitudes always amounts to 6 dB and the logarithmically transformed magnitude levels have equal differences. Hence, linear regression delivers the same slope for all three values mentioned above. The shapes of the derived equivalent sensation contours and the frequency-weighting curves depend only on the slope m of the calculated regression lines, not on the constant n (Table 5). One could hypothesize that the evaluation methods recommended in ISO 2631-1 do not correctly reflect the sensations. Therefore, it was supposed that the shape of the frequency-weighting curves derived from M1 to M3 differed from the W_d- curve. The equivalent intensity contours associated with the weighted accelerations M4 to M6 should deviate from a horizontal and straight line. If the assumption were true, the shape of the derived curves would reflect the deviation from the current evaluation methods.

Results

Step 1: Influence of repetition and time on the judgements

Differences between the judgements from the first and the second repetition were examined with the t-Test for paired samples (normal distribution, Kolmogorov-Smirnov-Test p=0.000 for all variables). No significant difference was found for the judgements of vibration intensity (p=0.122), but the judgements of comfort were significantly lower at the second repetition (p=0.001). Time effects on the reference signals were checked with Variance Analyses for repeated measures. There was no significant influence of time on the judgements (p≥0.165).

Table 4. Code of prediction method, basic functions, dependent and independent variables

Code of prediction	Function	Independent Variable	Dependent variable
A	power	a_{ov}	$LL_{original}$
B	power	a_{ov}	$LL_{norm,\ day}$
C	power	a_{ov}	$LL_{norm,\ all}$
D	linear	$lg(a_{ov})+c_1$	$lg(LL_{original})$
E	linear	$lg(a_{ov})+c_1$	$lg(LL_{norm,\ day}+c_2)+c_3$
F	linear	$lg(a_{ov})+c_1$	$lg(LL_{norm,\ all}+c_2)+c_3$

$LL_{original}$ – originally measured length of line in pixel, $LL_{norm,\ day}$ – length of line normalized with respect to individual means per experimental day, $LL_{norm,\ all}$ – length of line normalized with respect to individual means over all experimental days (normalized values in arbitrary units), a_{ov} – overall vibration total value, c_1, c_2, c_3 – constants for shifting values into positive ranges.

Step 2: Growth of sensation

According to Stevens' Law, the relation between physical stimulus and response can be described by a power function. Consequently, the relation between the logarithmically transformed stimuli and responses should be a linear function. Six prediction methods (see Table 4) were evaluated by comparing the coefficients of determination using (i) the original length of line and (ii) the normalized data with respect to individual means. The variables and functions are listed in Table 4. The parameters of the functions and the coefficients of determination were determined for each frequency, direction and type of vibration signal separately. In order to decide which model should be used for subsequent determination of equivalent sensation contours, the coefficients of determination of all frequencies, directions and types of vibration were organised according to the kind of function and range of definition. Figure 5 provides the mean values and confidence intervals of the coefficients of determination for the judgements of vibration intensity summarizing all frequencies, directions and types of vibration and divided as explained above.

The prediction models type E with the definition ranges M1–M6 n.w. and M1–M6 w. were used in the subsequent determinations of equivalent sensation contours as these models displayed the highest coefficients of determination (circled values in Fig. 5).

The values were shifted into positive ranges for the logarithmic transformation and the subsequent linear regression. Therefore, the minimum of $LL_{norm,day}$ of the entire data set was identified and used as c_2 (Table 4). Afterwards, the logarithmic transformation was performed. In the next step, the minima of $lg(a_{ov(n.w.)})$ and $lg(LL_{norm,day} + c_2)$ were detected and used as c_1 and c_3 (Table 4).

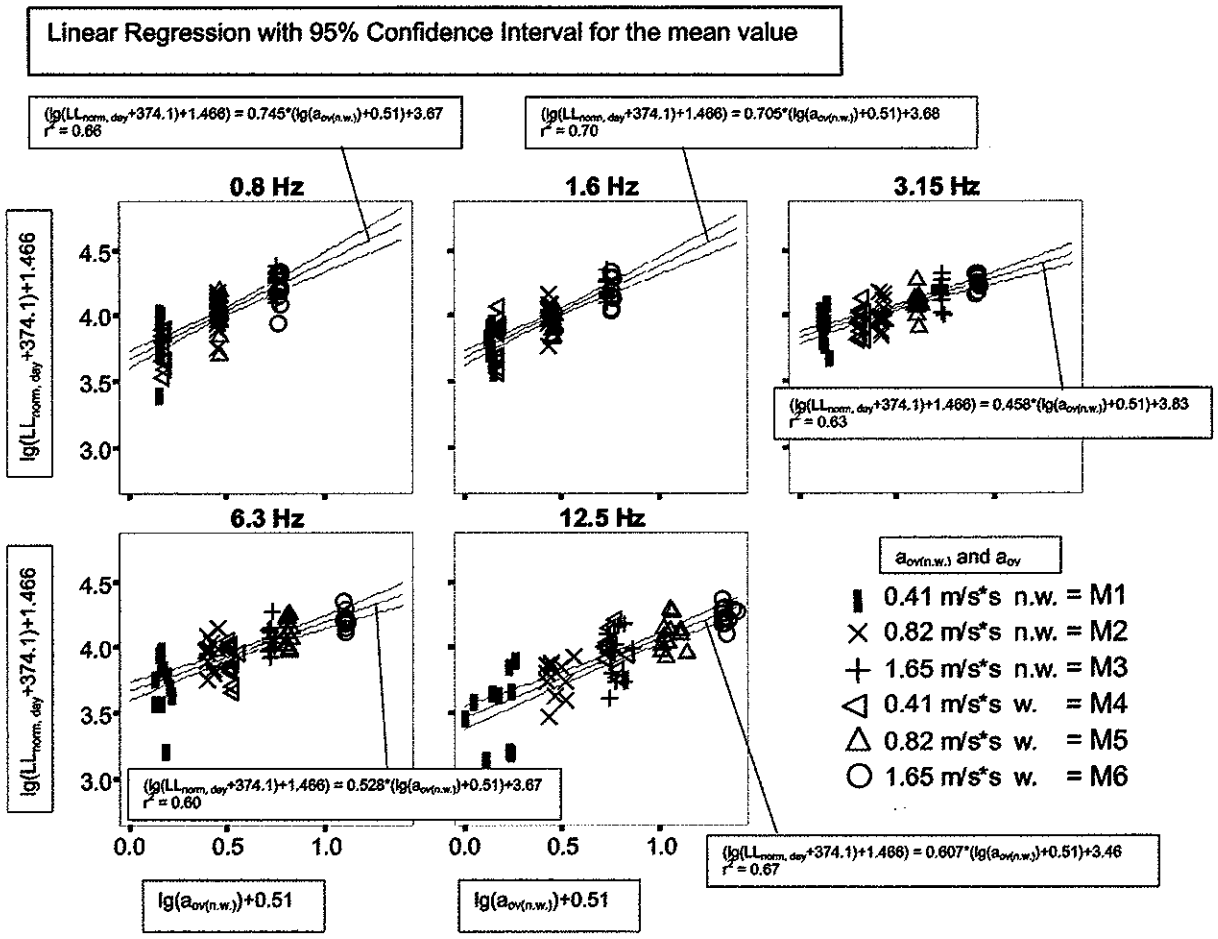

Fig. 6. Linear regression lines, slopes, constants and coefficients of determination for the prediction of judgements of vibration intensity for sinusoidal vibration, excitation in the x-axis, depending on the vibration frequency, range of definition M1–M6 n.w.

Table 5. Slope m, constant n and coefficient of determination r^2 of the linear regression

Type/Direction	Frequency [Hz]	Slope m	Constant n	Coefficient of determination r^2
Sinus X	0.8	0.745	3.67	0.66
	1.6	0.705	3.68	0.70
	3.15	0.458	3.83	0.63
	6.3	0.528	3.67	0.60
	12.5	0.607	3.46	0.67
Sinus Y	0.8	1.036	3.52	0.71
	1.6	0.901	3.61	0.75
	3.15	0.816	3.54	0.67
	6.3	0.565	3.62	0.67
	12.5	0.608	3.46	0.69
Band X	0.8	0.834	3.64	0.72
	1.6	0.784	3.71	0.76
	3.15	0.646	3.74	0.76
	6.3	0.504	3.65	0.56
	12.5	0.685	3.37	0.59
BandY	0.8	1.025	3.56	0.66
	1.6	0.819	3.71	0.79
	3.15	0.749	3.62	0.77
	6.3	0.648	3.55	0.60
	12.5	0.611	3.42	0.60

Equation (14) for the prediction of the judgement of vibration intensity.

Table 6. Slope m, constant n and coefficient of determination r^2 of the linear regression

Type/Direction	Frequency [Hz]	Slope	Constant	Coefficient of determination
Sinus X	0.8	–0.371	4.95	0.48
	1.6	–0.474	4.94	0.58
	3.15	–0.361	4.88	0.42
	6.3	–0.269	4.88	0.27
	12.5	–0.195	4.91	0.25
Sinus Y	0.8	–0.731	5.06	0.44
	1.6	–0.551	4.94	0.49
	3.15	–0.408	4.95	0.44
	6.3	–0.210	4.89	0.22
	12.5	–0.093	4.80	0.08
Band X	0.8	–0.497	4.91	0.53
	1.6	–0.493	4.84	0.52
	3.15	–0.424	4.82	0.38
	6.3	–0.253	4.82	0.35
	12.5	–0.182	4.85	0.27
Band Y	0.8	–0.763	4.96	0.50
	1.6	–0.633	4.82	0.33
	3.15	–0.490	4.85	0.36
	6.3	–0.319	4.88	0.27
	12.5	–0.142	4.83	0.23

Equation (16) for the prediction of the judgement of vibration comfort.

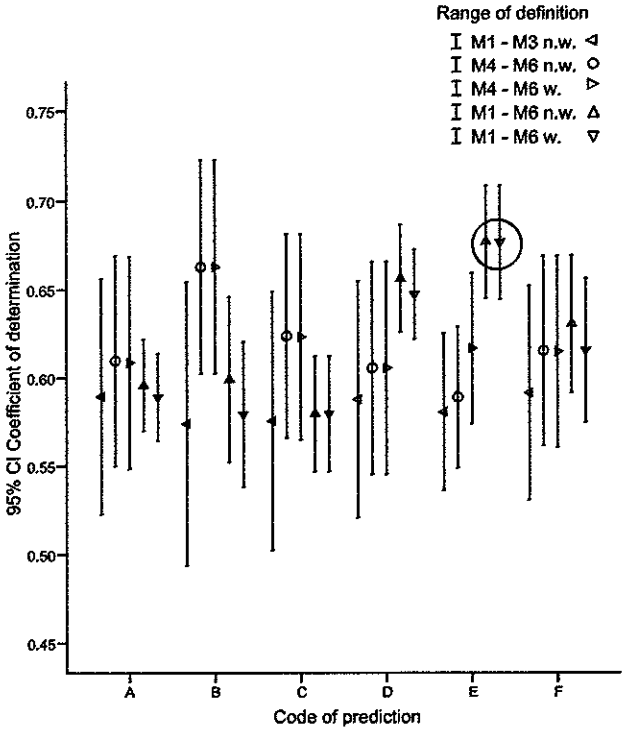

Fig. 5. Mean values and 95% confidence intervals (CI) of the coefficients of determination for the prediction of the judgements of vibration intensity summarizing all frequencies, directions and kinds of vibration, depending on the prediction method and on the range of definition.

The circle indicates the highest mean values of these coefficients of determination.

For the judgements of vibration intensity:

(14) Type E (M1–M6 n.w.):

$$(\lg(LL_{norm,day} + 374.1) + 1.466) = m \times (\lg(a_{ov(n.w.)}) + 0.51) + n$$

(15) Type E (M1–M6 w.):

$$(\lg(LL_{norm,day} + 374.1) + 1.466) = m \times (\lg(a_{ov}) + 1.01) + n$$

For the judgements of vibration comfort:

(16) Type E (M1–M6 n.w.):

$$(\lg(LL_{norm,day} + 428.1) + 2.1) = m \times (\lg(a_{ov(n.w.)}) + 0.51) + n$$

(17) Type E (M1–M6 w.):

$$(\lg(LL_{norm,day} + 428.1) + 2.1) = m \times (\lg(a_{ov}) + 1.01) + n$$

with

$LL_{norm,\ day}$ = length of line normalized with respect to individual means per experimental day

$a_{ov(n.w.)}$ = overall vibration total value, modified, non-weighted

a_{ov} = overall vibration total value, according ISO 2631-1, weighted

m = slope of the regression line

n = constant of the regression line

Figure 6 illustrates as an example the linear regression lines, equations with slopes, constants and coefficients of determination for the prediction of judgements of vibration intensity for sinusoidal vibration excitation in the x-axis (range of definition M1–M6 n.w.), depending on the vibration frequency. The parameters of the regression equations (14) and (16) for both types of signals and directions of excitation are listed in Table 5 (intensity, equation (14)) and Table 6 (comfort, equation (16)).

The coefficients of determination were much lower for the prediction of vibration comfort ($0.08 \le r^2 \le 0.58$) in particular for frequencies higher than 1.6 Hz ($0.08 \le r^2 \le 0.44$) (see Table 6).

Step 3: Equivalent vibration intensity and vibration comfort judgement contours

A model can be assumed to be sufficient when the coefficient of determination reaches a value of $r^2 = 0.5$ or more. Unfortunately, the prediction models for the comfort judgements had much lower coefficients (see Table 6). Therefore, equivalent sensation contours were derived only from the judgements of vibration intensity, not from the judgements of vibration comfort.

Equivalent intensity contours were determined by calculating the vibration acceleration corresponding to the intensity judgement at each frequency according to Equation (14) and Table 5, changing the range of value and the range of definition. Limits of the range of definition were taken into account when calculating the accelerations from the lengths of the lines. Therefore, the range of judgements (range of values) slightly varies between the figures for the different vibration directions and types (Figs. 7 and 8). The lowest and highest values were chosen so that the range of definition was completely filled but not exceeded at each frequency. The equivalent contours were then calculated in 12 steps of equidistant arbitrary units from the lowest to the highest equivalent contour. The equivalent intensity contours illustrate the vibration magnitudes required to produce the same strenght of sensation across the frequency range. They provide information on what frequencies produced greater sensation of intensity. A lower acceleration at a particular frequency indicates greater sensation of vibration intensity at that frequency. The overall shapes and the frequencies of highest sensitivity obviously depended on the magnitude, the direction and the type of vibration.

Figures 9 and 10 show ratios of predicted accelerations for frequencies above 0.8 Hz in relation to those at 0.8 Hz set to 1, and the inverted ratios in order to illustrate the effect of vibration magnitude on frequency weightings (Figs. 9 and 10). All values were multiplied with 1,000 in order to derive values comparable to those in ISO 2631-1 Table 3. The reference frequency $f_{ref} = 0.8$ Hz was selected as it was the frequency closest to that of highest sensitivity of the weighting curves W_d and $W_d \wedge W_k$ ($f_{sens} = 1.0$ Hz, see ISO 2631-1 Table 3 and Eq. (11)) and exposed in this study. Table 7 contains the values for sinusoidal excitation simultaneously exposed on the seat and the platform in the x-direction in 12 steps of equidistant arbitrary units (see Fig. 7(a) and Fig. 9 (a)). Additionally, the table encloses the magnitude independent factors for the W_d- and W_k-frequency weightings of ISO 2631-1 Table 3 and $W_d \wedge W_k$ (see Eq. (11)).

The following paragraph explains an example for the

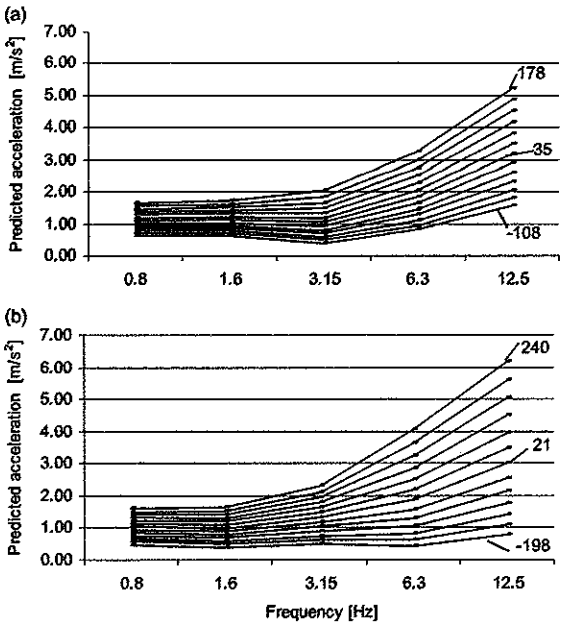

Fig. 7. Equivalent intensity contours for arbitrary sensation units from minimum to maximum of the range of value in order to meet the range of definition (range of actually exposed vibration magnitudes) in steps of 12 equidistant units determined from Eq. (14) and Table 5: (a) sinusoidal excitation in x-axis, (b) sinusoidal excitation in y-axis.

Predicted acceleration in [m/s^2] = overall vibration total value modified without frequency weighting $a_{ov(n.w.)}$ r.m.s.. Numbers attached to the lines: normalized intensity judgements $LL_{norm,\,day}$ (see Eq. (14)).

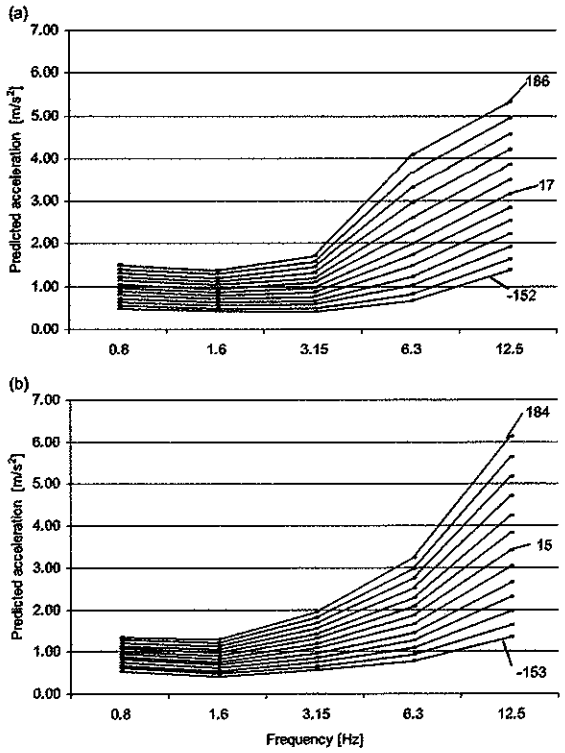

Fig. 8. Equivalent intensity contours for arbitrary sensation units from minimum to maximum of the range of value in order to meet the range of definition (range of actually exposed vibration magnitudes) in steps of 12 equidistant units determined from Eq. (14) and Table 5: (a) random octave band-width white noise excitation in x-axis, (b) random octave band-width white noise excitation in y-axis.

Predicted acceleration in [m/s^2] = overall vibration total value modified without frequency weighting $a_{ov(n.w.)}$ r.m.s..

Numbers attached to the lines: normalized intensity judgements $LL_{norm,\,day}$ (see Eq. (14)).

magnitude-dependence of the filter factors (2nd and 6th rows, marked in grey in Table 7). A sinusoidal vibration stimulus at 3.15 Hz with a magnitude of $a_{ov(n.w.)} = 0.49$ m/s^2 (0.69 m/s^2 × 0.716) produces a sensation equal to that of a sinusoidal vibration stimulus at 0.8 Hz with a magnitude of 0.69 m/s^2. In order to convert a measured sinusoidal vibration at 3.15 Hz with a magnitude of $a_{ov(n.w.)} = 0.49$ m/s^2 into a sinusoidal vibration stimulus at 0.8 Hz with equal intensity sensation, the acceleration has to be multiplied with 1.396 (filter factor), i.e. it has to be increased by 2.90 dB (20 × lg(1.396)). When the vibration signal has a magnitude of 0.93 m/s^2 (1.02 m/s^2 × 0.910) it has to be multiplied with 1.099 (filter factor), that means it has to be increased by 0.82 dB (20 × lg(1.099)) only.

Step 4: Equivalent intensity contours derived from weighted overall vibration total values

The equivalent intensity contours associated with the weighted accelerations a_{ov} were determined by the same method as described in step 3 but using Eq. (15). The slopes and constants are not given in detail. Assuming that the evaluation methods recommended in ISO 2631-1 correctly reflect the sensation, the equivalent intensity contours associated with the weighted accelerations a_{ov} should be horizontal and straight lines. But, they differed from straight lines. As expected from the results derived from the non-weighted accelerations, these contours reflected the differences between the contours obtained from the non-weighted accelerations $a_{ov(n.w.)}$ and the combination of the current weighting curves and multiplying factors W_d ^ W_k (Eq. (11)).

Discussion

Influence of repetition and time on the judgements

One experimental set lasted roughly two hours to two and a half hours. There were some doubts, whether the subjects were able to differentiate between the vibration comfort and the comfort of the entire situation including permanent demands on concentration and sitting a long period of time on the rigid seat without exercise. Schust[21] revealed that the subjects were not able to differentiate between the vibration comfort and the comfort of the entire situation when they had to judge the seat comfort. In Schust[21], the seat comfort decreased significantly with time. So, it could be the case that the vibration comfort judgements were influenced by time. Nevertheless, no significant decrease in comfort judgements of the identical reference stimuli per daily exposure set

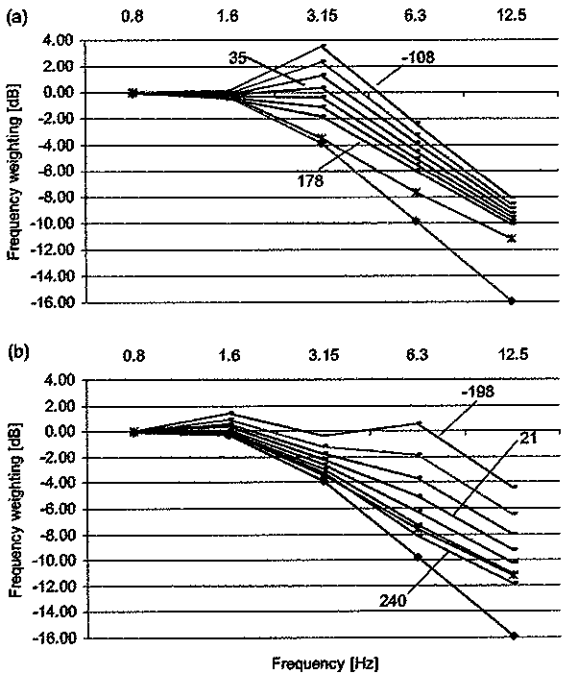

Fig. 9. Effect of vibration magnitude on frequency weightings (inverted equivalent intensity contours in steps of 6 equidistant arbitrary sensation units).

Numbers attached to the lines: normalized intensity judgements $LL_{norm,day}$ – length of line normalized with respect to individual means per experimental day. Curves normalized at 0.8 Hz and converted into dB: (a) sinusoidal excitation simultaneously exposed on the seat and the platform in x-direction, (b) sinusoidal excitation simultaneously exposed on the seat and the platform in y-direction. The results are compared with the frequency weightings.

W_d (–◆–) and $W_d \wedge W_k$ (–✳–) according to Eq. (11).

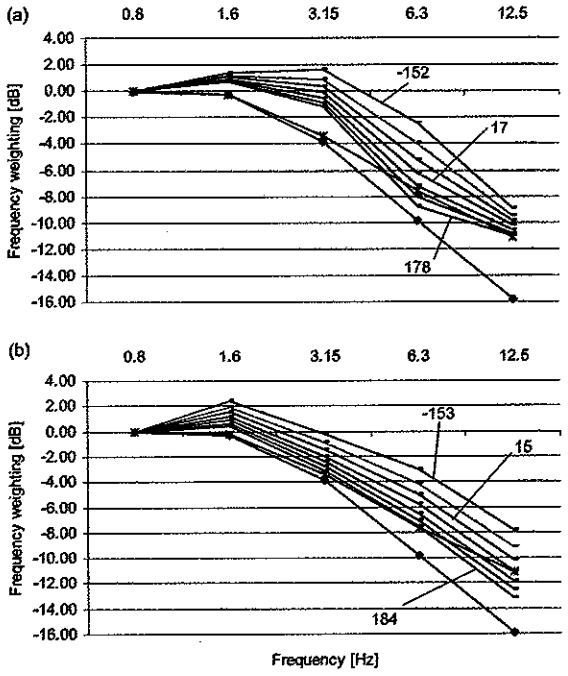

Fig. 10. Effect of vibration magnitude on frequency weightings (inverted equivalent intensity contours in steps of 6 equidistant arbitrary sensation units).

Numbers attached to the lines: normalized intensity judgements $LL_{norm,day}$ – length of line normalized with respect to individual means per experimental day. Curves normalized at 0.8 Hz and converted into dB: (a) octave band-width white noise excitation simultaneously exposed on the seat and the platform in x-direction, (b) octave band-width white noise excitation simultaneously exposed on the seat and the platform in y-direction.

The results are compared with the frequency weightings W_d (–◆–) and $W_d \wedge W_k$ (–✳–) according to Eq. (11).

was found. Moreover, the judgements of vibration intensity remained stable over time. A tendency to judge the very last exposure of the last experimental day less comfortable and more intensive was observed. Because of their four-day experience, the subjects knew that it was the very last exposure, even when it was not explicitly told them. That might be the reason for the slightly different judgement in comparison to the other reference stimuli. However, this tendency did not influence the general time independency of the judgements on one experimental day. The results suggest that the subjects were able to separate the intensity and comfort judgements from other perceptions associated with time effects.

On the other hand, the judgements of vibration comfort were significantly lower at the second repetition which was performed on a separate day. The reasons of this effect are not clear and so the interpretation is difficult. Possibly, the internal reference system concerning 'comfort' might vary from day to day. The topic is discussed more comprehensively in Section 1 and in the following paragraph. However, the results indicate an insufficient repeatability of the vibration comfort judgements.

Growth of sensation and equivalent sensation contours

The theory of cross-modality matching and the importance of Stevens' power law are described in Section 1. The Stevens' exponent was determined by regression analyses. The coefficients of determination for the prediction of comfort judgements were very low (see Table 6), so that these judgements supposed to be not suited for an adequate reflection of growth of sensation with vibration magnitude.

The interpretation of the outcomes relates to the term 'comfort'. As mentioned in Section 1, the term 'discomfort' does not exist in German. A simple inversion of the scale using the term 'comfort' does not seem to be a solution. Probably, 'comfort' is not just the opposite of 'discomfort'. 'Comfort' is rather associated with feelings of relaxation and well-being, whereas discomfort seems to be associated with biomechanical factors (joint angles, muscle contractions, pressure distribution) and tiredness (Zhang[22]). In a pilot study with 12 German speaking subjects (unpublished), the authors of the present investigation found that 'convenient' was the most appropriate word for 'comfortable', followed by cosy, pleasant, homelike, proper and easy. Therefore, the subjects were briefed to judge the vibration comfort bearing in mind all sensations related

Table 7. Ratios derived from equivalent vibration intensity contours dependending on the presented modified non-weighted overall vibration total value $a_{ov(n.w.)}$ at the reference frequency f_{ref} = 0.8 Hz ($a_{ov(n.w.),\ 0.8\ Hz}$) in case of sinusoidal excitation simultaneously presented on the seat and the platform in x-axis

		Ratios of predicted accelerations					Filter factors				
$a_{ov(n.w.)}$	f_{ref}	Ratio $a_{ov(n.w.)} / a_{ov(n.w.),\ 0.8\ Hz} \times 1{,}000$					Ratio $a_{ov(n.w.),\ 0.8\ Hz} / a_{ov(n.w.)} \times 1{,}000$				
		Frequency [Hz]					Frequency [Hz]				
		0.8	1.6	3.15	6.3	12.5	0.8	1.6	3.15	6.3	12.5
0.62	0.8	1,000	995	666	1,323	2,538	1,000	1,005	1,501	756	394
0.69	0.8	1,000	1,002	**716**	1,387	2,606	1,000	998	**1,396**	721	384
0.77	0.8	1,000	1,008	765	1,449	2,669	1,000	992	1,306	690	375
0.85	0.8	1,000	1,013	814	1,508	2,730	1,000	987	1,229	663	366
0.93	0.8	1,000	1,018	862	1,566	2,787	1,000	982	1,160	639	359
1.02	0.8	1,000	1,023	**910**	1,622	2,842	1,000	977	**1,099**	617	352
1.10	0.8	1,000	1,028	957	1,676	2,895	1,000	973	1,045	597	345
1.19	0.8	1,000	1,032	1,003	1,729	2,945	1,000	969	997	578	340
1.28	0.8	1,000	1,036	1,049	1,781	2,994	1,000	965	953	561	334
1.37	0.8	1,000	1,040	1,095	1,832	3,041	1,000	961	913	546	329
1.46	0.8	1,000	1,044	1,141	1,881	3,086	1,000	958	877	532	324
1.56	0.8	1,000	1,048	1,186	1,930	3,130	1,000	954	843	518	319
1.65	0.8	1,000	1,051	1,231	1,977	3,173	1,000	951	813	506	315
	f_{sens}						Factor × 1,000 (ISO 2631-1, Table 3)				
W_d	1.0	-	-	-	-	-	992	968	642	323	161
W_k	10.0	-	-	-	-	-	477	494	804	1,054	902
$W_d \wedge W_k$	1.0	-	-	-	-	-	999	976	673	417	277

Factors for W_d - and W_k - frequency weightings according to ISO 2631-1 Table 3 and for the combination of W_d - and W_k - factors and multiplying factors ($W_d \wedge W_k$) according to Eq. (11). Highest sensitivity of the weighting curve W_d and the combination $W_d \wedge W_k$: f_{sens} = 1.0 Hz. Highest sensitivity of the weighting curve W_k: f_{sens} = 10 Hz (see ISO 2631-1 Table 3 and Eq. (11)).

Table 8. Stevens' exponents for the growth of discomfort derived from magnitude estimation (Howarth[10] and Morioka[14]) and for the vibration intensity derived from cross-modality matching (present study) depending on the frequency and the direction of the exposed sinusoidal vibration

	Howarth[10]	Morioka[14]		Present study	
	y-direction	x-direction	y-direction	x-direction	y-direction
0.8				0.745	1.036
1.6				0.705	0.901
2		0.948	0.635		
2.5		0.668	0.763		
3.15		***0.499***	***0.742***	***0.458***	***0.816***
4	0.68	0.461	0.932		
5		0.468	0.876		
5.6	0.85				
6.3		0.805	0.953	0.528	0.565
8	0.93	0.711	0.716		
10		0.735	0.935		
11.3	1.41				
12.5		0.854	0.907	0.607	0.608

to these terms. Moreover, they were requested to ignore the surrounding influences like climate, noise, demands on concentration and whether the mouse could easily be gripped or not. Notwithstanding this, the vibration comfort judgements seemed to be affected by many influences in addition to the vibration magnitude.

In contrast, coefficients of determination for the prediction of intensity judgements were high enough to presume the linear model to be appropriate for an adequate reflection of growth of sensation with increasing vibration magnitude (see Table 5). It was of interest to see whether the obtained exponents were similar to those derived from discomfort judgements reported by other authors. There are some comparable investigations with horizontal excitations of the seat or simultaneously of the seat and at the feet. Morioka[14] and Howarth[10] reported Stevens' exponents determined by magnitude estimation in their studies (Table 8).

For reasons discussed in the next paragraph, only the exponents derived at 3.15 Hz in the present study were reasonably comparable to those from Morioka[14] at the same frequency (in bold characters in Table 8). In spite of different methods, these exponents are very similar. In all three studies, Stevens'

exponents varied within the frequency range, and frequency dependent Stevens' exponents cause magnitude dependent equivalent sensation contours.

In order to compare the results to those from other authors and to reveal a possibly systematic influence of relative body movements on the outcomes, some studies with horizontal vibrations were divided into investigations with and without relative body movements in the following paragraphs.

Griffin[7], Howarth[10] and Morioka[14] performed studies with vibration at the seat only, with stationary feet and hands.

Griffin[7] reported an experiment which determined the levels of fore-and-aft and lateral seat vibrations at seven frequencies (1, 2, 4, 8, 16, 31.5 and 63 Hz) causing discomfort equivalent to 0.5 and 1.25 m/s^2 r.m.s. 10 Hz vertical seat vibration. The vibration magnitudes of the test motions varied from 0.1 to 20 m/s^2. The subjects' feet were not vibrated and there was no backrest. Over the investigated range of levels the differences in equivalent sensation contours were small. The authors concluded that it seems reasonable to determine and apply a single equivalent comfort contour. They did not directly compare their results to the frequency weightings described in standards.

Howarth[10] exposed the subjects to six acceleration levels of sinusoidal vibrations in the y- and z-axes in a very low range from 0.04 m/s^2 to 0.4 m/s^2 at nine frequencies between 4 and 63 Hz. The footrest was stationary and there was no backrest. The authors found a magnitude dependence of the equivalent sensation contours. However, the frequency weightings were averaged over six magnitudes and compared with Wd frequency weighting defined in BSI 6841 (1987)[23] and ISO 2631 (1985)[24]. It was concluded that the averaged frequency weightings for sinusoidal vibration in the y-axis were in good agreement with W_d over the whole frequency range.

In experiments performed by Morioka[14], the subjects judged the discomfort caused by sinusoidal vibration in all three directions at frequencies between 2 and 315 Hz. The magnitudes varied from minimum 0.02 m/s^2 to maximum 1.25 m/s^2 r.m.s. in 3 dB steps. The range of exposed magnitude levels increased with increasing frequency in order to ensure that the stimuli were above the perception thresholds but not likely to be considered excessively unpleasant. There was no backrest and stationary handles and footrests were used. There were some magnitude dependent differences between the derived equivalent sensation contours and the W_d-curve, more pronounced for vibrations in the x-direction than in the y-direction (Fig. 8 in Morioka[14]).

Donati[25] and Corbridge[9] performed studies with identical exposure on the seat and at the feet.

Donati[25] compared the subjective response of seated subjects to sinusoidal vibrations in x-, y- and z-axes in the 1–10 Hz range with those produced by narrow-band random vibration centred at the same frequencies using the 'floating reference vibration' method. The accelerations varied from about 0.6 m/s^2 to about 4.0 m/s^2. The subjects sat on a semi-rigid seat or a rigid seat with and without support by a backrest. Identical vibrations were exposed simultaneously on the seat, the feet (footrest) and the hands (steering wheel). The differences between ISO-weighting and equivalent sensation contours were comparable with those obtained in the present study (Fig. 10 in[25]). The authors concluded that the equivalent sensation contours derived from these experiments related only roughly to the weighting curves in ISO 2631-1, particularly in the x-direction. The magnitude dependence of weighting curves was not systematically investigated in this study. Corbridge[9] conducted experiments with lateral sinusoidal vibration in the 0.5–5.0 Hz range. The magnitudes varied from 0.4 to 3.15 m/s^2. Subjects were seated on a rigid wooden seat and rested their feet on the moving vibrator table. The seat had a flat backrest, but the authors did not exactly describe whether it was used or not. The authors concluded that the experimentally determined contours for lateral vibration were in reasonable agreement with the curve defined in ISO 2631 (1978) (Fig. 9 in[9]). In both studies it remained vague whether the comparisons were related to the W_d- curves or to a combination of multiplying factors and W_d- and W_k-curves because of the simultaneous exposure of seat and feet.

Discussing the influence of relative body movements on the outcomes, one could suppose an increase of vibration sensitivity at least at low frequencies when only the seat was excited. When comparing the results of these studies with the outcomes of investigations with simultaneous vibration on the seat and the feet, a systematic difference at least at low frequencies may be expected. However, both types of experimental design delivered evidence varying from reasonable agreement with the ISO-curves to obvious differences without any systematic divergences. The relative body movements might influence the sensations less than assumed. In experiments with horizontal vibrations (modified signals of mobile machines) and professional driver seats with fixed or activated horizontal suspension, Schust[26] revealed high correlations between judgements of vibration intensity and vibration magnitude but only weak to middle correlations between intensity judgements and movements of the head in the room and the angle velocity of the bending of the trunk, the latter for exposures in y-direction and some exposure conditions only. The authors concluded that the subjective judgement of the intensity seems to depend rather on the vibration magnitude at the buttocks, the back and the feet than on the movements of the body parts in relation to the space coordinates or the relative movements between the body parts. Moreover, in this study twenty different values of acceleration were calculated for analyses of correlation between accelerations and subjective judgements, amongst others the point vibration total value av and the overall vibration total value a_{ov}. Comparing the results for av and aov the authors assumed the vibration measuring point (platform, seat or backrest) to be probably of minor importance for the association between acceleration and judgement of intensity, at least for the exposure conditions tested in this study with no extensively relative movements between these points.

Moreover, there is a lack of literature concerning an exact explanation of methods which were used to derive the frequency-weighting curves and the guide for their application with regard to health, comfort and perception (ISO 2631-1, Table 1) including the multiplying factors k (ISO 2631-1, clauses 7 and 8).

Bearing in mind the facts discussed above, it is difficult to decide whether equivalent sensation contours for the seat should be (i) derived from experiments with excitation on the seat only or with simultaneous exposure on the seat and the

Table 9. Predicted accelerations depending on arbitrary sensation units and on frequency derived by Morioka[14] (first two rows) and in the present study (last seven rows)

	Frequency [Hz]										
Arbitrary units	0.8	1.6	2	2.5	3.15	4	5	6.3	8	10	12.5
50			0.073	0.062	0.055	0.057	0.071	0.195	0.226	0.308	0.421
25			0.041	0.030	0.023	0.023	0.026	0.091	0.101	0.145	0.219
–108	0.62	0.62			0.41			0.82			1.57
–84	0.69	0.70			0.50			0.96			1.81
–60	0.77	0.78			0.59			1.12			2.06
–36	0.85	0.86			0.69			1.29			2.33
–13	0.93	0.95			0.80			1.46			2.60
11	1.02	1.04			0.93			1.65			2.89
35	1.10	1.13			1.06			1.85			3.19

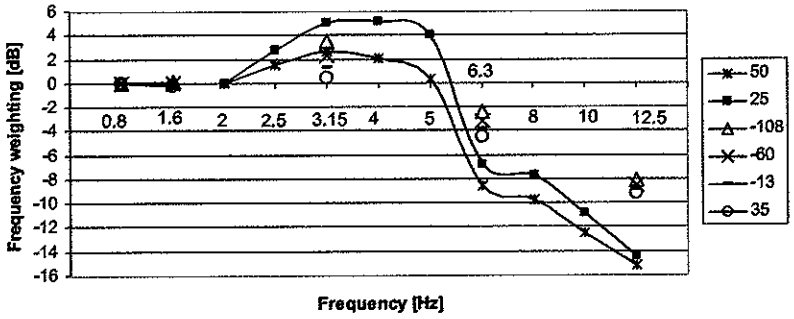

Fig. 11. Frequency weightings for vibration exposure in x-direction depending on arbitrary sensation units derived by Morioka (arbitrary units 25 and 50) and in the present study (arbitrary units –108, –60, –13 and 35).

feet, (ii) derived from the point overall vibration total value or the overall vibration total value (a_v or a_{ov}) and (iii) compared with W_d or $W_d \wedge W_k$.

In the present study, due to technical reasons an excitation merely on the seat was not realizable. That means there was a simultaneous exposure to vibration on the seat and at the feet. Moreover, the subjects were briefed to judge integratively the entire vibration exposure. Therefore, it was supposed that the judgements were reflected more likely by the overall vibration total value a_{ov} than by the point vibration total value av or the vibration in the axis of excitation only. Consequently, the relations between the a_{ov} and the subjective judgements were determined by curve fitting and the obtained equivalent sensation contours were discussed mainly in comparison with $W_d \wedge W_k$. In all Figures with frequency weightings, the W_d -curve is also given. At low frequencies both curves do not differ considerably but at frequencies from 6.3 Hz upwards they diverge by more than 2.2 dB and from 8 Hz upwards they diverge by more than 3 dB due to the effect of WBV acting on the feet. Assuming the current evaluation methods using the weighted overall vibration value a_{ov} adequately reflect the sensations, these differences were surely detectable by the subjects. It might be the case that, the effect of the WBV acting on the feet, when the seat and the feet are simultaneously excited, restricts the examination of the frequency-weighting curve W_d at frequencies above 5 Hz third-octave band mid frequency.

Comparing the results of the present study with these from Morioka[14], there seems to be some evidence for this assumption. Both studies are similar. Morioka[14] also investigated sinusoidal vibration, but no random excitation. The frequencies of standardisation for the frequency-weighting curves differed because of the different lowest frequencies (2 Hz in Morioka[14], 0.8 Hz in the present study). Moreover, there were only 3 common frequencies investigated (3.15, 6.3 and 12.5 Hz) and the magnitudes at the frequency of normalization were much more lower in Morioka[14] (0.041 m/s^2 to 0.417 m/s^2 r.m.s. in x- direction, 0.02 m/s^2 to 0.63 m/s^2 r.m.s. in y-direction) compared with the present study (0.41 m/s^2 to 1.70 m/s^2 r.m.s. in x- and y-directions, see Table 1). However, almost identical Stevens' exponents were obtained for frequencies at 3.15 Hz (see Table 8). Above 3.15 Hz, Morioka's exponents are higher, which indicates a deeper slope of the frequency-weighting curve.

For sinusoidal excitation in y-direction, similar filter factors for 3.15 Hz were derived when similar magnitudes were used at the frequency which was used for normalization (e.g. 0.6 m/s^2 at 2 Hz in Morioka[14] and at 0.8 Hz in the present study). However, above 3.15 Hz Morioka's curves are closer to W_d than the frequency weightings obtained in the present investigation. That might be due to separate vibration of the seat. Morioka[14] found similar shapes for the frequency-weighting curves for sinusoidal excitation in x-direction with the highest sensitivity around 2–3.15 Hz, but for lower vibration magnitudes (0.041 m/s^2 to 0.073 m/s^2 r.m.s. at 2 Hz) compared with the present investigation (0.62 m/s^2 to 1.19 m/s^2 r.m.s. at 0.8 Hz) (Table 9). Figure 11 shows the frequency weightings for vibration exposure in x-direction

depending on arbitrary sensation units derived by Morioka (arbitrary units 25 and 50) and in the present study (arbitrary units –108, –60, –13 and 35).

One could be tempted to extrapolate the data to make some studies comparable by exceeding the range of definition but that would assume a questionable linearity in the human response. For instance, Miwa[26] reported a reduction in the exponent with increasing vibration magnitude.

Some differences in the results might be due to different judgement methods. The present investigation seems to be only one which used cross-modality matching. The method has the advantage not to be dependent on a 'vibration memory', which means that the subjects do not have to keep in mind the sensation regarding a previously exposed reference stimulus in order to judge the current stimulus. On the other hand, cross-modality matching takes more time because of the necessity of a pre-period for vibration sensation (about 20 s) before judging the stimulus. Therefore, the number of conditions (magnitudes, frequencies etc.) realizable in an experimental session is restricted in order not to exceed an acceptable duration.

The weighted magnitude levels M4, M5 and M6 had overall vibration total values numerically equal to the non-weighted magnitudes M1, M2, M3. Therefore, the experiment was performed de facto twice, once with non-weighted magnitudes and repeatedly with weighted values. The multiplying factors were used for calculating both, M1 to M3 and M4 to M6. Assuming that the evaluation methods recommended in ISO 2631-1 correctly reflect the sensations, one could hypothesize that the shape of the frequency-weighting curves derived from M1 to M3 reflected the W_d- curve or the W_d ^ W_k –curve and the equivalent intensity contours associated with the weighted accelerations M4 to M6 were horizontal and straight lines. If the assumption were not true, the shape of the derived curves would reflect the deviation from the current evaluation methods. The latter was the case (see Fig. 12).

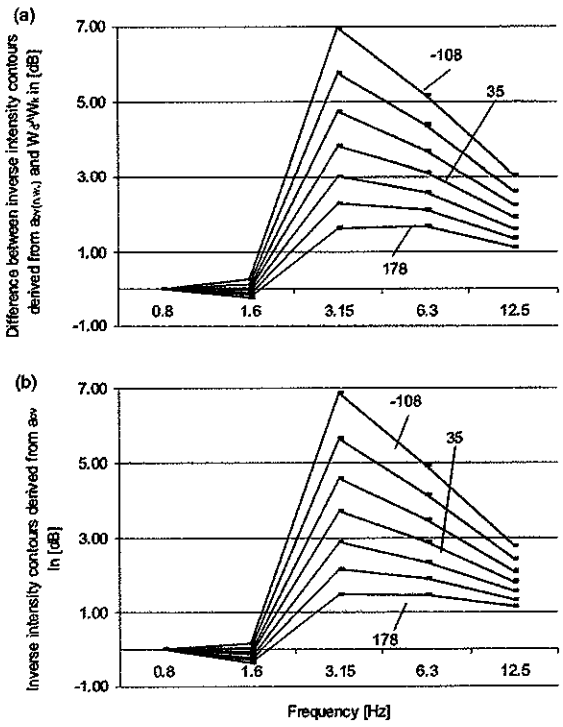

Fig. 12. (a) Difference between inverse intensity contours depending on arbitrary sensation units in 6 equidistant steps, derived from modified non-weighted overall vibration total values $a_{ov(n.w.)}$ (referred to 0.8 Hz) and W_d ^ W_k in dB for sinusoidal excitation x-direction (see Fig. 9 (a)) (b) Inverse intensity contours derived from weighted overall vibration total values a_{ov} (referred to 0.8 Hz) in dB.
All curves depending on arbitrary sensation units in 6 equidistant steps.

Conclusions

The differences between the obtained equivalent intensity contours and the current frequency weightings according to ISO 2631-1 were the following:

- strong dependency on vibration magnitude
- underestimation of the sensation varying in extent from 2 dB to 8 dB at 1.6, 3.15, 6.3 and 12.5 Hz in comparison with the reference frequency 0.8 Hz for all signals, with the most pronounced effects revealed at the frequencies 3.15 and 6.3 Hz and at lower intensities (a_{ov} around 0.48 m/s^2 to 0.8 m/s^2 r.m.s. at the reference frequency 0.8 Hz).
- some differences in the frequency weightings for sinusoidal and random octave band-width signals which should not be overinterpreted because of the restricted number of exposure conditions investigated in the study

The limitations of the study are the following:

- The study design did not allow differentiation between the frequency weightings W_d and W_k and the multiplying factor 0.25.
- Only five frequencies and six magnitude levels were investigated in order not to exceed an acceptable duration of a daily session.
- There was no multi-axis vibration.

The research on frequency weightings is currently not at a stage to be transferable for use in practice. More effort is needed to investigate the effects of vibrations typical for mobile workplaces, in particular for cases of multi-axis vibration. Moreover, further investigations should try to tackle the problem of evaluation of combined vibration at different input positions and relative movements between the body parts. Although it is commonly supposed that the sensation is a prerequisite for adverse health effects, there are doubts whether the findings from studies using subjective judgements are applicable, for instance, to the prediction of spinal injuries. The association between vibration signals weighted with altered filter factors and health effects should be confirmed.

Acknowledgement

The authors thank Dr. N. Gizem Forta for proof-reading the paper.

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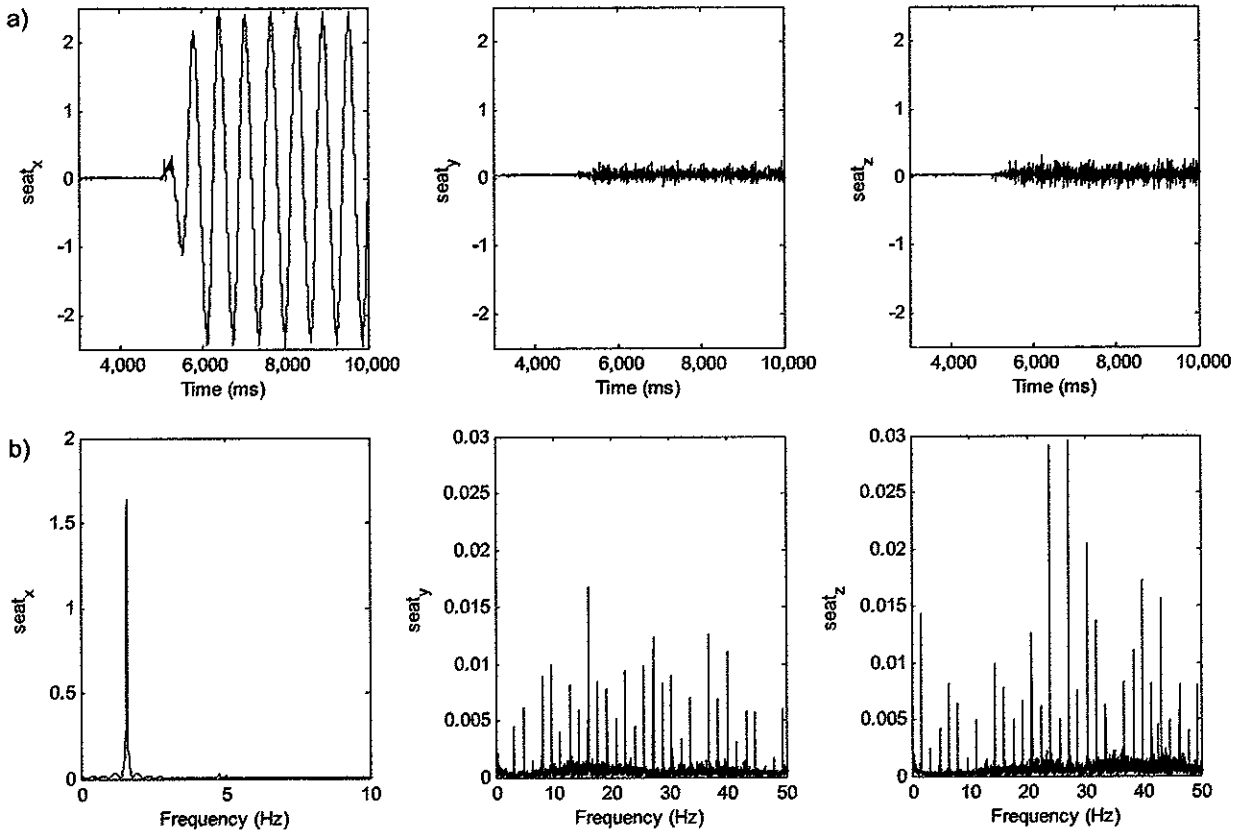
Appendix A

Example of measured non-weighted acceleration in m/s^2 at the seat in x-, y- and z-axes. Subject 36, sinusoidal excitation in x-direction, magnitude M3 (r.m.s. $a_{des,ex,seat}$ = 1.6 m/s^2), frequency F2 (1.6 Hz), repetition 1.
a) time signals (3rd to 10th second: 2 s pre-period + 1 s exposure with tapering + 4 s exposure).
b) FFT-analyses (6th to 69th second: 63 s exposure without tapering) (see also Fig. 2).



Appendix B: Instructions for the experiment

General information
You will sit down on a rigid seat and fasten the seat belt, which is not to be opened without a request by the operator. During the experiment, you will be exposed to vibration. The motions of the simulator will be monitored for the entire duration of the experiment. Minor deviations from the desired motions will lead to deactivation of the device. You will be able to shut the simulator down by using the emergency stop button. After switching off, the platform moves down slowly. During this process, the platform may temporarily remain in an inclined position. You will perceive different vibrations. The test conditions will vary and will be presented in random order. At certain times you will be asked to judge the intensity and the comfort of the vibration. Please follow the instructions given on the screen.

Judgements
A line will appear on the screen shortly after a question. The line will automatically become longer or shorter.

You should try to adjust the length of the line in accordance with your sensation, using the mouse buttons:
The *stronger* the sensation - the *longer* the line.

You can stop the extension and the shortening of the line with the right or the left mouse button. You can adjust the length of the line with the mouse buttons as well. Pressing the right button shortens the line, pressing the left button lengthens it. You can confirm the chosen length with a double click on the middle mouse button (the scroll wheel). Please tell the operator when you have been restricted due to the maximum length of the presented line.
You will be asked to judge the following sensations:

How intensive do you perceive the vibration to be?
This means the intensity of the vibration. Please concentrate on the vibration and disregard all additional influences such as noise, temperature, air quality, illumination or the comfort of the vibration. The latter will be judged separately.
The more intensive the vibration - the longer the line.

How comfortable do you perceive the vibration to be?
This means sensations which may relate to the comfort of vibration, for example sensations that you would associate with a convenient, cosy, pleasant, homely, proper etc. state.

Please, judge the experimental conditions only regarding the vibration comfort, and ignore, for example, the temperature, air quality, noise, accessibility of the mouse or the demands for your attention during the judgement.
The more comfortable the vibration - the longer the line.

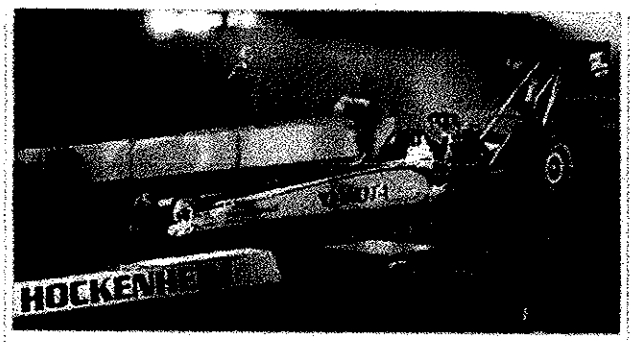
R

R

g-force

From Wikipedia, the free encyclopedia

The **g-force** (with *g* from *gravitational*) associated with an object is its acceleration relative to free-fall.[1][2] This acceleration experienced by an object is due to the vector sum of non-gravitational forces acting on an object free to move. The accelerations that are not produced by gravity are termed proper accelerations, and it is only these that are measured in g-force units. They cause stresses and strains on objects, which are felt as weight (any g-force can thus be simply described, and measured, as a "weight per unit mass"). Because of these strains (weight forces), large proper accelerations (large g-forces), may be destructive.



This top-fuel dragster can accelerate from zero to 160 kilometres per hour (100 mph) in 0.86 seconds. This is a horizontal acceleration of 5.3 g. Combined with the vertical g-force in the stationary case the Pythagorean theorem yields a g-force of 5.4 g.

The standard gravitational acceleration at the Earth's surface produces g-force only indirectly. The 1 g force on an object sitting on the Earth's surface is caused by mechanical force exerted in the upward direction by the ground, keeping the object from going into free-fall. An object on the Earth's surface is accelerating relative to the free-fall condition, which is the path an object would follow falling freely toward the Earth's center. It is thus experiencing proper acceleration, even without a change in velocity (which is dv/dt, the familiar "coordinate acceleration" of Newton's laws).

Objects allowed to free-fall under the influence of gravity feel no g-force, as demonstrated by the "zero-g" conditions inside a freely-falling elevator falling toward the Earth's center (in vacuum), or (to good approximation) conditions inside a spacecraft in Earth orbit. These are examples of coordinate acceleration (a change in velocity) *without* proper acceleration. Since the g-force felt is always a measure of proper acceleration (which, in these cases, is zero, even though the objects are freely changing velocity due to gravity) all of these conditions of free-fall produce no g-force. The experience of no g-force (zero-g), however it is produced, is synonymous with weightlessness.

In the absence of gravitational fields, or in directions at right angles to them, proper and coordinate accelerations are the same, and any coordinate acceleration must be produced by a corresponding g-force acceleration. An example here is a rocket in free space, in which simple changes in velocity are produced by the engines, and produce g-forces on the rocket and passengers. The same happens in a dragster (see illustration) when it is changing velocity in a direction at right angles to the acceleration of gravity: such changes must be produced by accelerations that are appropriately measured in g-force units in the horizontal direction, since they produce g-force effects in that direction.

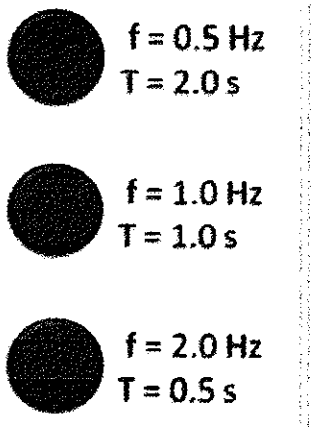
Contents

S

Hertz

From Wikipedia, the free encyclopedia

The **hertz** (symbol: **Hz**) is the SI unit of frequency defined as the number of cycles per second of a periodic phenomenon.[1] One of its most common uses is the description of the sine wave, particularly those used in radio and audio applications.



Lights flash at *frequency* f = 0.5 Hz (Hz = hertz), 1.0 Hz and 2.0 Hz, where *x* Hz means *x* flashes per second. T is the *period* and T = *y* s (s = second) means that *y* is the number of seconds per flash. T and f are each other's reciprocal: f = 1/T and T = 1/f.

Contents

Definition

The hertz is equivalent to cycles per second.[2] In defining the second the CIPM declared that "the standard to be employed is the transition between the hyperfine levels F = 4, M = 0 and F = 3, M = 0 of the ground state $2S_{1/2}$ of the caesium 133 atom, unperturbed by external fields, and that the frequency of this transition is assigned the value 9 192 631 770 hertz"[3] thereby effectively defining the hertz and the second simultaneously.

In English, hertz is used as a plural. As an SI unit, Hz can be prefixed; commonly used multiples are kHz (kilohertz, 10^3 Hz), MHz (megahertz, 10^6 Hz), GHz (gigahertz, 10^9 Hz) and THz (terahertz, 10^{12} Hz). One hertz simply means "one cycle per second" (typically that which is being counted is a complete cycle); 100 Hz means "one hundred cycles per second", and so on. The unit may be applied to any periodic event—for example, a clock might be said to tick at 1 Hz, or a human heart might be said to beat at 1.2 Hz. The "frequency" (activity) of aperiodic or stochastic events, such as radioactive decay, is expressed in becquerels.

Even though angular velocity, angular frequency and hertz all have the dimensions of *1/s*, angular velocity and angular frequency are *not* expressed in hertz,[4] but rather in an appropriate angular unit such as radians per second. Thus a disc rotating at 60 revolutions per minute (rpm) is said to be rotating at either

Hertz	
Unit system:	SI derived unit
Unit of...	Frequency
Symbol:	Hz

Named after:	Heinrich Hertz
In SI base units:	1 Hz = 1/s

2π rad/s *or* 1 Hz, where the former measures the angular velocity and latter reflects the number of *complete* revolutions per second. The conversion between a frequency f measured in hertz and an angular velocity ω measured in radians per second are:

$$\omega = 2\pi f \text{ and } f = \omega/(2\pi).$$

This SI unit is named after Heinrich Hertz. As with every SI unit whose name is derived from the proper name of a person, the first letter of its symbol is upper case (**Hz**). When an SI unit is spelled out in English, it should always begin with a lower case letter (**hertz**), except where *any* word would be capitalized, such as at the beginning of a sentence or in capitalized material such as a title. Note that "degree Celsius" conforms to this rule because the "d" is lowercase. —Based on *The International System of Units (http://www.bipm.org/en/si/si_brochure/chapter5/5-2.html)* , section 5.2.

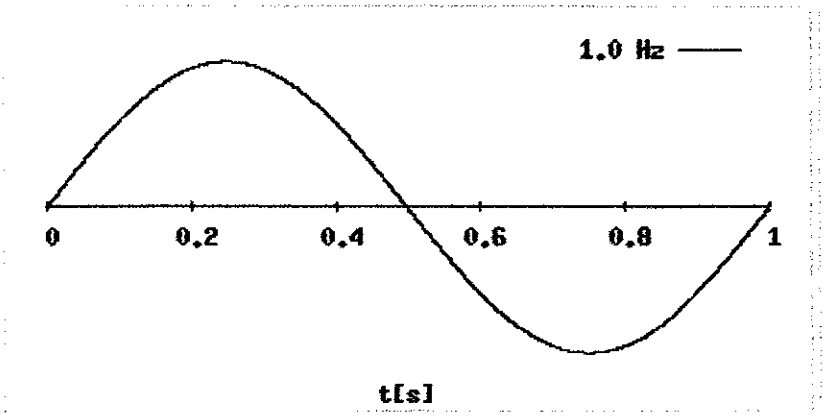
History

The hertz is named after the German physicist Heinrich Hertz, who made important scientific contributions to the study of electromagnetism. The name was established by the International Electrotechnical Commission (IEC) in 1930.[5] It was adopted by the General Conference on Weights and Measures (CGPM) (*Conférence générale des poids et mesures*) in 1960, replacing the previous name for the unit, *cycles per second* (cps), along with its related multiples, primarily *kilocycles per second* (kc/s) and *megacycles per second* (Mc/s), and occasionally *kilomegacycles per second* (kMc/s). The term *cycles per second* was largely replaced by *hertz* by the 1970s.

The term "gigahertz", most commonly used in computer processor clock rates and radio frequency (RF) applications, can be pronounced either /ˈgɪgəhɜrts/, with a hard /g/ sound, or /ˈdʒɪgəhɜrts/, with a soft /dʒ/.[6] The prefix "giga-" is derived directly from the Greek "γιγας."

Applications

Vibration

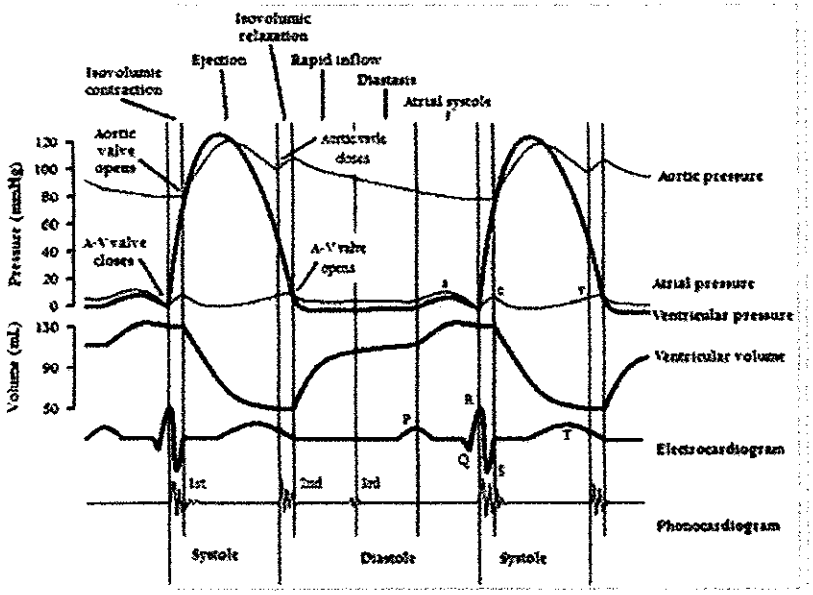


Waves of different frequency.

Sound is a traveling wave which is an oscillation of pressure. Humans perceive frequency of sound waves as pitch. Each musical note corresponds to a particular frequency which can be measured in hertz. An infant's ear is able to perceive frequencies ranging from 20 Hz to 20,000 Hz; the average adult human can hear sounds between 20 Hz and 16,000 Hz.[7] The range of ultrasound, infrasound and other physical vibrations such as molecular vibrations extends into the megahertz range and well beyond.

Electromagnetic radiation

Electromagnetic radiation is often described by its frequency—the number of oscillations of the perpendicular electric and magnetic fields per second—expressed in hertz.



Details of a heartbeat as an example of a non-sinusoidal periodic phenomenon that can be described in terms of hertz. Two complete cycles are illustrated.

Radio frequency radiation is usually measured in kilohertz, megahertz, or gigahertz; this is why radio dials are commonly labeled with kHz, MHz, and GHz. Light is electromagnetic radiation that is even higher in frequency, and has frequencies in the range of tens (infrared) to thousands (ultraviolet) of terahertz. Electromagnetic radiation with frequencies in the low terahertz range, (intermediate between those of the highest normally usable radio frequencies and long-wave infrared light), is often called terahertz radiation. Even higher frequencies exist, such as that of gamma rays, which can be measured in exahertz. (For historical reasons, the frequencies of light and higher frequency electromagnetic radiation are more commonly specified in terms of their wavelengths or photon energies: for a more detailed treatment of this and the above frequency ranges, see electromagnetic spectrum.)

Computing

In computing, most central processing units (CPU) are labeled in terms of their clock rate expressed in megahertz or gigahertz (10^9 hertz). This number refers to the frequency of the CPU's master clock signal ("Clock rate"). This signal is simply an electrical voltage which changes from low to high and back again at regular intervals. This signal is also referred to as a square wave. Hertz has become the primary unit of measurement accepted by the general populace to determine the performance of a CPU, but many experts have criticized this approach, which they claim is an easily manipulable benchmark.[8] For home-based personal computers, the CPU has ranged from approximately 1 megahertz in the late 1970s (Atari, Commodore, Apple computers) to up to 6 GHz in the present (IBM POWER processors).

Various computer buses, such as the front-side bus connecting the CPU and northbridge, also operate at different frequencies in the megahertz range (for modern products).

CRT television and monitor refresh rates are measured in hertz.

SI multiples

SI multiples for hertz (Hz)

Submultiples			Multiples		
Value	**Symbol**	**Name**	**Value**	**Symbol**	**Name**
10^{-1} Hz	dHz	decihertz	10^{1} Hz	daHz	decahertz
10^{-2} Hz	cHz	centihertz	10^{2} Hz	hHz	hectohertz
10^{-3} Hz	mHz	millihertz	10^{3} Hz	**kHz**	**kilohertz**
10^{-6} Hz	μHz	microhertz	10^{6} Hz	**MHz**	**megahertz**
10^{-9} Hz	nHz	nanohertz	10^{9} Hz	**GHz**	**gigahertz**
10^{-12} Hz	pHz	picohertz	10^{12} Hz	**THz**	**terahertz**
10^{-15} Hz	fHz	femtohertz	10^{15} Hz	PHz	petahertz
10^{-18} Hz	aHz	attohertz	10^{18} Hz	EHz	exahertz
10^{-21} Hz	zHz	zeptohertz	10^{21} Hz	ZHz	zettahertz
10^{-24} Hz	yHz	yoctohertz	10^{24} Hz	YHz	yottahertz
Common prefixed units are in bold face.					

Frequencies not expressed in hertz

Even higher frequencies are believed to occur naturally, in the frequencies of the quantum-mechanical wave functions of high-energy (or, equivalently, massive) particles, although these are not directly observable, and must be inferred from their interactions with other phenomena. For practical reasons, these are typically not expressed in hertz, but in terms of the equivalent quantum energy, which is proportional to the frequency by the factor of Planck's constant.

See also

- Alternating current
- Electronic tuner
- Frequency changer
- Normalized frequency
- Orders of magnitude (frequency)
- Radian per second
- Signal bandwidth

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3. ^ "[Resolutions of the (http://www.bipm.org/utils/common/pdf/si_brochure_8_en.pdf) CIPM, 1964 - Atomic and molecular frequency standards"]. SI brochure, Appendix 1. http://www.bipm.org/utils/common/pdf/si_brochure_8_en.pdf. Retrieved 2010-20-26.
4. ^ "SI brochure, Section 2.2.2, paragraph 6" (http://www.bipm.org/en/si/derived_units/2-2-2.html) . http://www.bipm.org/en/si/derived_units/2-2-2.html.

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6. ^ "Gigahertz (http://dictionary.reference.com/browse/gigahertz) " in Dictionary.com Unabridged. *Dictionary*.
7. ^ Dominant spectral region (http://www.mmk.e-technik.tu-muenchen.de/persons/ter/top/dominant.html)
8. ^ Good Riddance, Gigahertz (http://www.wired.com/news/business/0,1367,62851,00.html)

External links

- BIPM Cesium ion f_{Cs} definition (http://www.bipm.org/en/si/si_brochure/chapter2/2-1/second.html)
- National Research Council of Canada: *Generation of the Hz* (http://inms-ienm.nrc-cnrc.gc.ca/research/frequency_time_projects_e.html#gen)
- National Research Council of Canada: *Cesium fountain clock* (http://inms-ienm.nrc-cnrc.gc.ca/research/cesium_clock_e.html)
- National Physical Laboratory: *Trapped ion optical frequency standards* (http://www.npl.co.uk/server.php?show=ConWebDoc.1086)
- National Research Council of Canada: *Optical frequency standard based on a single trapped ion* (http://inms-ienm.nrc-cnrc.gc.ca/research/optical_frequency_projects_e.html#optical)
- National Research Council of Canada: *Optical frequency comb* (http://inms-ienm.nrc-cnrc.gc.ca/research/optical_frequency_projects_e.html#femtosecond)
- One Hertz in Radians per Second (Google). Note, as of 06 May 2009 there is an error of 2π. (http://www.google.com/search?q=one+hertz+in+radians+per+second)

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ICHD-2

Main article: International Classification of Headache Disorders

The International Classification of Headache Disorders (ICHD) is an in-depth hierarchical classification of headaches published by the International Headache Society. It contains explicit (operational) diagnostic criteria for headache disorders. The first version of the classification, ICHD-1, was published in 1988. The current revision, ICHD-2, was published in 2004.[6]

The classification uses numeric codes. The top, one-digit diagnostic level includes 14 headache groups. The first four of these are classified as primary headaches, groups 5-12 as secondary headaches, cranial neuralgia, central and primary facial pain and other headaches for the last two groups.[7]

NIH

Main article: NIH classification of headaches

The NIH classification consists of brief definitions of a limited number of headaches.[2]

Symptoms and signs

Headache associated with specific symptoms may warrant urgent medical attention, particularly sudden, severe headache or sudden headache associated with a stiff neck; headaches associated with fever, convulsions or accompanied by confusion or loss of consciousness; headaches following a blow to the head, or associated with pain in the eye or ear; persistent headache in a person with no previous history of headaches; and recurring headache in children.

Pathophysiology

The brain in itself is not sensitive to pain, because it lacks nociceptors. However, several areas of the head and neck do have nociceptors, and can thus sense pain. These include the extracranial arteries, large veins, cranial and spinal nerves, head and neck muscles and the meninges.[8]

Diagnosis

In 2008, the American College of Emergency Physicians updated their guidelines on the evaluation and management of adult patients who have a nontraumatic headache of acute onset.[8]

While, statistically, headaches are most likely to be primary (harmless and self-limiting), some specific secondary headache syndromes may demand specific treatment or may be warning signals of more serious disorders. Differentiating between primary and secondary headaches can be difficult.

As it is often difficult for patients to recall the precise details regarding each headache, it is often useful for the sufferer to fill-out a "headache diary" detailing the characteristics of the headache.

Imaging

When the headache does not clearly fit into one of the recognized primary headache syndromes or when atypical symptoms or signs are present then further investigations are justified.[9] Neuroimaging (noncontrast head CT) is recommended if there are new neurological problems such as decreased level of consciousness, one sided weakness, pupil size difference, etc or if the pain is of sudden onset and severe, or if the person is known HIV positive.[8] People over the age of 50 years may also warrant a CT scan.[8]

Treatment

Acute headaches

Not all headaches require medical attention, and most respond with simple analgesia (painkillers) such as paracetamol/acetaminophen or members of the NSAID class (such as aspirin/acetylsalicylic acid, diclofenac or ibuprofen).

A small 2009 study found that 100% oxygen at 15 l / min was effective at relieving undifferentiated headache pain in the emergency department.[10]

Chronic headaches

See also: Management of chronic headaches

In recurrent unexplained headaches keeping a "headache diary" with entries on type of headache, associated symptoms, precipitating and aggravating factors may be helpful. This may reveal specific patterns, such as an association with medication, menstruation or absenteeism or with certain foods. It was reported in March 2007 by two separate teams of researchers that stimulating the brain with implanted electrodes appears to help ease the pain of cluster headaches.[11]

Acupuncture has been found to be beneficial in chronic headaches[12] of both tension type[13] and migraine type.[14] Whether or not there is a difference between true acupuncture and sham acupuncture however is yet to be determined.[14]

Epidemiology

During a given year, 90% of people suffer with headaches. Of the ones who are seen in the ER, about 1% have a serious underlying problem.[15]

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External links

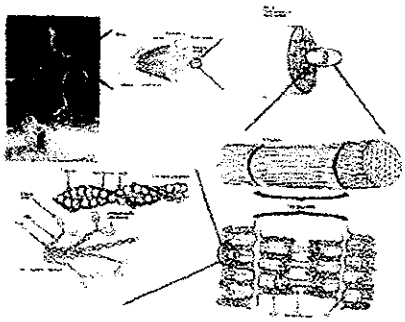
- National Headache Foundation
- IHS - The International Headache Classification (ICHD-2)
- American Headache Society
- Withdrawal related headache information

Retrieved from "http://en.wikipedia.org/wiki/Headache"
Categories: Pain | Headaches | Neurological disorders | Symptoms

Muscle

From Wikipedia, the free encyclopedia

Muscle (from Latin *musculus*, diminutive of *mus* "mouse"[1]) is the contractile tissue of animals and is derived from the mesodermal layer of embryonic germ cells. Muscle cells contain contractile filaments that move past each other and change the size of the cell. They are classified as skeletal, cardiac, or smooth muscles. Their function is to produce force and cause motion. Muscles can cause either locomotion of the organism itself or movement of internal organs. Cardiac and smooth muscle contraction occurs without conscious thought and is necessary for survival. Examples are the contraction of the heart and peristalsis which pushes food through the digestive system. Voluntary contraction of the skeletal muscles is used to move the body and can be finely controlled. Examples are movements of the eye, or gross movements like the quadriceps muscle of the thigh. There are two broad types of voluntary muscle fibers: slow twitch and fast twitch. Slow twitch fibers contract for long periods of time but with little force while fast twitch fibers contract quickly and powerfully but fatigue very rapidly.



A top-down view of skeletal muscle

Muscles are predominately powered by the oxidation of fats and carbohydrates, but anaerobic chemical reactions are also used, particularly by fast twitch fibers. These chemical reactions produce adenosine triphosphate (ATP) molecules which are used to power the movement of the myosin heads.

Contents

Embryology

All muscles derive from paraxial mesoderm. The paraxial mesoderm is divided along the embryo's length into somites, corresponding to the segmentation of the body (most obviously seen in the vertebral column. Each somite has 3 divisions, sclerotome (which forms vertebrae), dermatome (which forms skin), and myotome (which forms muscle). The myotome is divided into two sections, the epimere and hypomere, which form epaxial and hypaxial muscles, respectively. Epaxial muscles in humans are only the erector spinae and small intervertebral muscles, and are innervated by the dorsal rami of the spinal nerves. All other muscles, including limb muscles, are hypaxial muscles, formed from the hypomere, and inervated by the ventral rami of the spinal nerves.

During development, myoblasts (muscle progenitor cells) either remain in the somite to form muscles associated with the vertebral column or migrate out into the body to form all other muscles. Myoblast migration is preceded by the formation of connective tissue frameworks, usually formed from the somatic lateral plate mesoderm. Myoblasts follow chemical signals to the appropriate locations, where they fuse into elongate skeletal muscle cells.

Types

There are three types of muscle:

Types of muscle (shown at different magnifications)

- Skeletal muscle or "voluntary muscle" is anchored by tendons (or by aponeuroses at a few places) to bone and is used to effect skeletal movement such as locomotion and in maintaining posture. Though this postural control is generally maintained as a subconscious reflex, the muscles responsible react to conscious control like non-postural muscles. An average adult male is made up of 42% of skeletal muscle and an average adult female is made up of 36% (as a percentage of body mass).[2]
- Smooth muscle or "involuntary muscle" is found within the walls of organs and structures such as the esophagus, stomach, intestines, bronchi, uterus, urethra, bladder, blood vessels, and the arrector pili in the skin (in which it controls erection of body hair). Unlike skeletal muscle, smooth muscle is not under conscious control.
- Cardiac muscle is also an "involuntary muscle" but is more akin in structure to skeletal muscle, and is found only in the heart.

Cardiac and skeletal muscles are "striated" in that they contain sarcomeres and are packed into highly-regular arrangements of bundles; smooth muscle has neither. While skeletal muscles are arranged in regular, parallel bundles, cardiac muscle connects at branching, irregular angles (called intercalated discs). Striated muscle contracts and relaxes in short, intense bursts, whereas smooth muscle sustains longer or even near-permanent contractions.

Skeletal muscle is further divided into several subtypes:

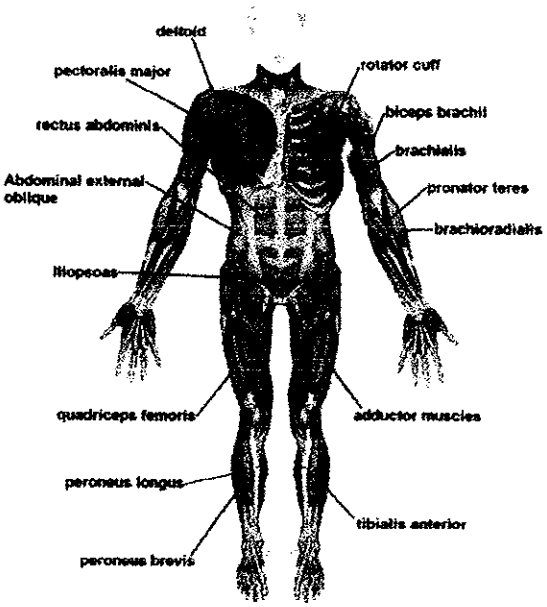
- Type I, slow oxidative, slow twitch, or "red" muscle is dense with capillaries and is rich in mitochondria and myoglobin, giving the muscle tissue its characteristic red color. It can carry more oxygen and sustain aerobic activity.
- Type II, fast twitch muscle, has three major kinds that are, in order of increasing contractile speed:[3]
  - Type IIa, which, like slow muscle, is aerobic, rich in mitochondria and capillaries and appears red.
  - Type IIx (also known as type IId), which is less dense in mitochondria and myoglobin. This is the fastest muscle type in humans. It can contract more quickly and with a greater amount of force than oxidative muscle, but can sustain only short, anaerobic bursts of activity before muscle contraction becomes painful (often incorrectly attributed to a build-up of lactic acid). N.B. in some books and articles this muscle in humans was, confusingly, called type IIB.[4]
  - Type IIb, which is anaerobic, glycolytic, "white" muscle that is even less dense in mitochondria and myoglobin. In small animals like rodents this is the major fast muscle type, explaining the pale color of their flesh.

Anatomy

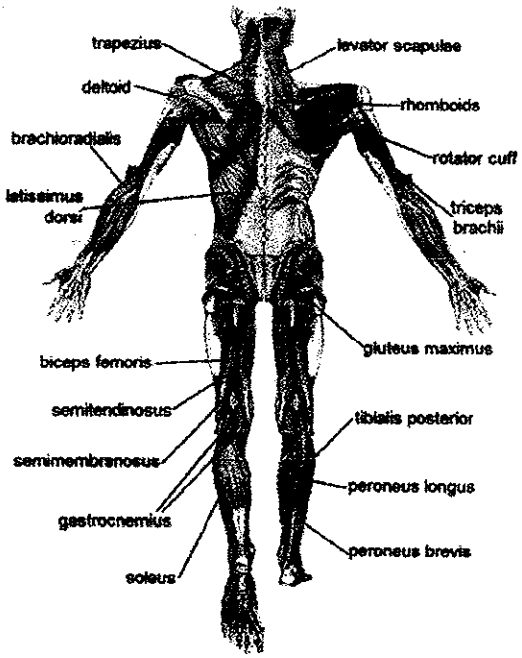
The anatomy of muscles includes both gross anatomy, comprising all the muscles of an organism, and, on the other hand, microanatomy, which comprises the structures of a single muscle.

Gross anatomy

The gross anatomy of a muscle is the most important indicator of its role in the body. The action a muscle generates is determined by the origin and insertion locations. The cross-sectional area of a muscle (rather than volume or length) determines the amount of force it can generate by defining the number of sarcomeres which can operate in parallel. The amount of force applied to the external environment is determined by lever mechanics, specifically the ratio of in-lever to out-lever. For example, moving the insertion point of the biceps more distally on the radius (farther from the joint of rotation) would increase the force generated during flexion (and, as a result, the maximum weight lifted in this movement), but decrease the maximum speed of flexion. Moving the insertion point proximally (closer to the joint of rotation) would result in decreased force but increased velocity. This can be most easily seen by comparing the limb of a mole to a horse - in the former, the insertion point is positioned to maximize force (for digging), while in the latter, the insertion point is positioned to maximize speed (for running).



Muscles, anterior view (*See Gray's muscle pictures for detailed pictures)*



Muscles, posterior view (*See Gray's muscle pictures for detailed pictures)*

One particularly important aspect of gross anatomy of muscles is pennation or lack thereof. In most muscles, all the fibers are oriented in the same direction, running in a line from the origin to the insertion. In pennate muscles, the individual fibers are oriented at an angle relative to the line of action, attaching to the origin and insertion tendons at each end. Because the contracting fibers are pulling at an angle to the overall action of the muscle, the change in length is smaller, but this same orientation allows for more fibers (thus more force) in a muscle of a given size. Pennate muscles are usually found where their length change is less important than maximum force, such as the rectus femoris.

There are approximately 639 skeletal muscles in the human body. However, the exact number is difficult to define because different sources group muscles differently.

Main article: Table of muscles of the human body

Microanatomy

Muscle is mainly composed of muscle cells. Within the cells are myofibrils; myofibrils contain sarcomeres, which are composed of actin and myosin. Individual muscle fibres are surrounded by endomysium. Muscle fibers are bound together by perimysium into bundles called fascicles; the bundles are then grouped together to form muscle, which is enclosed in a sheath of epimysium. Muscle spindles are distributed throughout the muscles and provide sensory feedback information to the central nervous system.

Skeletal muscle is arranged in discrete muscles, an example of which is the *biceps brachii*. It is connected by tendons to processes of the skeleton. Cardiac muscle is similar to skeletal muscle in both composition and action, being comprised of myofibrils of sarcomeres, but anatomically different in that the muscle fibers are typically branched like a tree and connect to other cardiac muscle fibers through intercalcated discs, and form the appearance of a syncytium.

Physiology

Main article: muscle contraction

The three types of muscle (skeletal, cardiac and smooth) have significant differences. However, all three use the movement of actin against myosin to create contraction. In skeletal muscle, contraction is stimulated by electrical impulses transmitted by the nerves, the motor nerves and motoneurons in particular. Cardiac and smooth muscle contractions are stimulated by internal pacemaker cells which regularly contract, and propagate contractions to other muscle cells they are in contact with. All skeletal muscle and many smooth muscle contractions are facilitated by the neurotransmitter acetylcholine.

Muscular activity accounts for much of the body's energy consumption. All muscle cells produce adenosine triphosphate (ATP) molecules which are used to power the movement of the myosin heads. Muscles conserve energy in the form of creatine phosphate which is generated from ATP and can regenerate ATP when needed with creatine kinase. Muscles also keep a storage form of glucose in the form of glycogen. Glycogen can be rapidly converted to glucose when energy is required for sustained, powerful contractions. Within the voluntary skeletal muscles, the glucose molecule can be metabolized anaerobically in a process called glycolysis which produces two ATP and two lactic acid molecules in the process (note that in aerobic conditions, lactate is not formed; instead pyruvate is formed and transmitted through the citric acid cycle). Muscle cells also contain globules of fat, which are used for energy during aerobic exercise. The aerobic energy systems take longer to produce the ATP and reach peak efficiency, and requires many more biochemical steps, but produces significantly more ATP than anaerobic glycolysis. Cardiac muscle on the other hand, can readily consume any of the three macronutrients (protein, glucose and fat) aerobically without a 'warm up' period and always extracts the maximum ATP yield from any molecule involved. The heart, liver and red blood cells will also consume lactic acid produced and excreted by skeletal muscles during exercise.

Nervous control

Efferent leg

The efferent leg of the peripheral nervous system is responsible for conveying commands to the muscles and glands, and is ultimately responsible for voluntary movement. Nerves move muscles in response to voluntary and autonomic (involuntary) signals from the brain. Deep muscles, superficial muscles, muscles of the face and internal muscles all correspond with dedicated regions in the primary motor cortex of the brain, directly anterior to the central sulcus that divides the frontal and parietal lobes.

In addition, muscles react to reflexive nerve stimuli that do not always send signals all the way to the brain. In this case, the signal from the afferent fiber does not reach the brain, but produces the reflexive movement by direct connections with the efferent nerves in the spine. However, the majority of muscle activity is volitional, and the result of complex interactions between various areas of the brain.

Nerves that control skeletal muscles in mammals correspond with neuron groups along the primary motor cortex of the brain's cerebral cortex. Commands are routed though the basal ganglia and are modified by input from the cerebellum before being relayed through the pyramidal tract to the spinal cord and from there to the motor end plate at the muscles. Along the way, feedback, such as that of the extrapyramidal system contribute signals to influence muscle tone and response.

Deeper muscles such as those involved in posture often are controlled from nuclei in the brain stem and basal ganglia.

Afferent leg

The afferent leg of the peripheral nervous system is responsible for conveying sensory information to the brain, primarily from the sense organs like the skin. In the muscles, the muscle spindles convey information about the degree of muscle length and stretch to the central nervous system to assist in maintaining posture and joint position. The sense of where our bodies are in space is called proprioception, the perception of body awareness. More easily demonstrated than explained, proprioception is the "unconscious" awareness of where the various regions of the body are located at any one time. This can be demonstrated by anyone closing their eyes and waving their hand around. Assuming proper proprioceptive function, at no time will the person lose awareness of where the hand actually is, even though it is not being detected by any of the other senses.

Several areas in the brain coordinate movement and position with the feedback information gained from proprioception. The cerebellum and red nucleus in particular continuously sample position against movement and make minor corrections to assure smooth motion.

Exercise

Exercise is often recommended as a means of improving motor skills, fitness, muscle and bone strength, and joint function. Exercise has several effects upon muscles, connective tissue, bone, and the nerves that stimulate the muscles.

Various exercises require a predominance of certain muscle fiber utilization over another. Aerobic exercise involves long, low levels of exertion in which the muscles are used at well below their maximal contraction strength for long periods of time (the most classic example being the marathon). Aerobic events, which rely primarily on the aerobic (with oxygen) system, use a higher percentage of Type I (or slow-twitch) muscle fibers, consume a mixture of fat, protein and carbohydrates for energy, consume large amounts of oxygen and produce little lactic acid. Anaerobic exercise involves short bursts of higher intensity contractions at a much greater percentage of their maximum contraction strength. Examples of anaerobic exercise include sprinting and weight lifting. The anaerobic energy delivery system uses predominantly Type II or fast-twitch muscle fibers, relies mainly on ATP or glucose for fuel,

consumes relatively little oxygen, protein and fat, produces large amounts of lactic acid and can not be sustained for as long a period as aerobic exercise. The presence of lactic acid has an inhibitory effect on ATP generation within the muscle; though not producing fatigue, it can inhibit or even stop performance if the intracellular concentration becomes too high. However, long-term training causes neovascularization within the muscle, increasing the ability to move waste products out of the muscles and maintain contraction. Once moved out of muscles with high concentrations within the sarcomere, lactic acid can be used by other muscles or body tissues as a source of energy, or transported to the liver where it is converted back to pyruvate. The ability of the body to export lactic acid and use it as a source of energy depends on training level.

Humans are genetically predisposed with a larger percentage of one type of muscle group over another. An individual born with a greater percentage of Type I muscle fibers would theoretically be more suited to endurance events, such as triathlons, distance running, and long cycling events, whereas a human born with a greater percentage of Type II muscle fibers would be more likely to excel at anaerobic events such as a 200 meter dash, or weightlifting.

Delayed onset muscle soreness is pain or discomfort that may be felt one to three days after exercising and subsides generally within two to three days later. Once thought to be caused by lactic acid buildup, a more recent theory is that it is caused by tiny tears in the muscle fibers caused by eccentric contraction, or unaccustomed training levels. Since lactic acid disperses fairly rapidly, it could not explain pain experienced days after exercise.[5]

Muscular, spinal and neural factors all affect muscle building. Sometimes a person may notice an increase in strength in a given muscle even though only its opposite has been subject to exercise, such as when a bodybuilder finds her left biceps stronger after completing a regimen focusing only on the right biceps. This phenomenon is called cross education.

Disease

Main article: Neuromuscular disease

Symptoms of muscle diseases may include weakness, spasticity, myoclonus and myalgia. Diagnostic procedures that may reveal muscular disorders include testing creatine kinase levels in the blood and electromyography (measuring electrical activity in muscles). In some cases, muscle biopsy may be done to identify a myopathy, as well as genetic testing to identify DNA abnormalities associated with specific myopathies and dystrophies.

Neuromuscular diseases are those that affect the muscles and/or their nervous control. In general, problems with nervous control can cause spasticity or paralysis, depending on the location and nature of the problem. A large proportion of neurological disorders leads to problems with movement, ranging from cerebrovascular accident (stroke) and Parkinson's disease to Creutzfeldt-Jakob disease.

A non-invasive elastography technique that measures muscle noise is undergoing experimentation to provide a way of monitoring neuromuscular disease. The sound produced by a muscle comes from the shortening of actomyosin filaments along the axis of the muscle. During contraction, the muscle shortens along its longitudinal axis and expands across the transverse axis, producing vibrations at the surface.[6]

Atrophy

Main article: Muscle atrophy

There are many diseases and conditions which cause a decrease in muscle mass, known as muscle atrophy. Examples include cancer and AIDS, which induce a body wasting syndrome called cachexia. Other syndromes or conditions which can induce skeletal muscle atrophy are congestive heart disease and some diseases of the liver.

During aging, there is a gradual decrease in the ability to maintain skeletal muscle function and mass, known as sarcopenia. The exact cause of sarcopenia is unknown, but it may be due to a combination of the gradual failure in the "satellite cells" which help to regenerate skeletal muscle fibers, and a decrease in sensitivity to or the availability of critical secreted growth factors which are necessary to maintain muscle mass and satellite cell survival. Sarcopenia is a normal aspect of aging, and is not actually a disease state yet can be linked to many injuries in the elderly population as well as decreasing quality of life[7].

Physical inactivity and atrophy

Inactivity and starvation in mammals lead to atrophy of skeletal muscle, accompanied by a smaller number and size of the muscle cells as well as lower protein content.[8] In humans, prolonged periods of immobilization, as in the cases of bed rest or astronauts flying in space, are known to result in muscle weakening and atrophy. Such consequences are also noted in small hibernating mammals like the golden-mantled ground squirrels and brown bats.[9]

Bears are an exception to this rule; species in the family Ursidae are famous for their ability to survive unfavorable environmental conditions of low temperatures and limited nutrition availability during winter by means of hibernation. During that time, bears go through a series of physiological, morphological and behavioral changes.[10] Their ability to maintain skeletal muscle number and size at time of disuse is of a significant importance.

During hibernation, bears spend four to seven months of inactivity and anorexia without undergoing muscle atrophy and protein loss.[9] There are a few known factors that contribute to the sustaining of muscle tissue. During the summer period, bears take advantage of the nutrition availability and accumulate muscle protein. The protein balance at time of dormancy is also maintained by lower levels of protein breakdown during the winter time.[9] At times of immobility, muscle wasting in bears is also suppressed by a proteolytic inhibitor that is released in circulation.[8] Another factor that contributes to the sustaining of muscle strength in hibernating bears is the occurrence of periodic voluntary contractions and involuntary contractions from shivering during torpor.[11] The three to four daily episodes of muscle activity are responsible for the maintenance of muscle strength and responsiveness in bears during hibernation.[11]

Strength

A display of "strength" (e.g. lifting a weight) is a result of three factors that overlap: **physiological strength** (muscle size, cross sectional area, available crossbridging, responses to training), **neurological strength** (how strong or weak is the signal that tells the muscle to contract), and **mechanical strength** (muscle's force angle on the lever, moment arm length, joint capabilities). Contrary to popular belief, the number of muscle fibres cannot be increased through exercise; instead the muscle cells simply get bigger. Muscle fibres have a limited capacity for growth through hypertrophy and some believe they split through hyperplasia if subject to increased demand.

The "strongest" human muscle

Since three factors affect muscular strength simultaneously and muscles never work individually, it is misleading to compare strength in individual muscles, and state that one is the "strongest". But below are several muscles whose strength is noteworthy for different reasons.

- In ordinary parlance, muscular "strength" usually refers to the ability to exert a force on an external object—for example, lifting a weight. By this definition, the masseter or jaw muscle is the strongest. The 1992 Guinness Book of Records records the achievement of a bite strength of 4,337 N (975 lb_f) for 2 seconds. What distinguishes the masseter is not anything special about the muscle itself, but its advantage in working against a much shorter lever arm than other muscles.
- If "strength" refers to the force exerted by the muscle itself, e.g., on the place where it inserts into a bone, then the strongest muscles are those with the largest cross-sectional area. This is because the tension exerted by an individual skeletal muscle fiber does not vary much. Each fiber can exert a force on the order of 0.3 micronewton. By this definition, the strongest muscle of the body is usually said to be the quadriceps femoris or the gluteus maximus.
- A shorter muscle will be stronger "pound for pound" (i.e., by weight) than a longer muscle. The myometrial layer of the uterus may be the strongest muscle by weight in the human body. At the time when an infant is delivered, the entire human uterus weighs about 1.1 kg (40 oz). During childbirth, the uterus exerts 100 to 400 N (25 to 100 lbf) of downward force with each contraction.
- The external muscles of the eye are conspicuously large and strong in relation to the small size and weight of the eyeball. It is frequently said that they are "the strongest muscles for the job they have to do" and are sometimes claimed to be "100 times stronger than they need to be." However, eye movements (particularly saccades used on facial scanning and reading) do require high speed movements, and eye muscles are exercised nightly during rapid eye movement sleep.
- The statement that "the tongue is the strongest muscle in the body" appears frequently in lists of surprising facts, but it is difficult to find any definition of "strength" that would make this statement true. Note that the tongue consists of sixteen muscles, not one.
- The heart has a claim to being the muscle that performs the largest quantity of physical work in the course of a lifetime. Estimates of the power output of the human heart range from 1 to 5 watts. This is much less than the maximum power output of other muscles; for example, the quadriceps can produce over 100 watts, but only for a few minutes. The heart does its work continuously over an entire lifetime without pause, and thus does "outwork" other muscles. An output of one watt continuously for eighty years yields a total work output of two and a half gigajoules.

Efficiency

The efficiency of human muscle has been measured (in the context of rowing and cycling) at 18% to 26%.[12] The efficiency is defined as the ratio of mechanical work output to the total metabolic cost, as can be calculated from oxygen consumption. This low efficiency is the result of about 40% effiency of generating ATP from food energy, losses in converting energy from ATP into mechanical work inside the muscle, and mechanical losses inside the body. The latter two losses are dependent on the type of exercise and the type of muscle fibers being used (fast-twitch or slow-twitch). For an overal efficiency of 20 percent, one watt of mechanical power is equivalent to 4.3 kcal per hour. For example, a manufacturer of rowing equipment shows burned calories as four times the actual mechanical work, plus 300 kcal per hour,[13] which amounts to about 20 percent efficiency at 250 watts of mechanical output.

Density of muscle tissue compared to adipose tissue

The density of mammalian skeletal muscle tissue is about 1.06 kg/liter[14]. This can be contrasted with the density of adipose tissue (fat), which is 0.9196 kg/liter[15]. This makes muscle tissue approximately 15% denser than fat tissue.

Muscle evolution

Evolutionarily, specialized forms of skeletal and cardiac muscles predated the divergence of the vertebrate/arthropod evolutionary line.[16] This indicates that these types of muscle developed in a common ancestor sometime before 700 million years ago (mya). Vertebrate smooth muscle was found to have evolved independently from the skeletal and cardiac muscles.

See also

- Atrophy
- Bodybuilding
- Cross education
- Electroactive polymers (materials that behave like muscles, used in robotics research)
- Fascia
- Hand strength
- List of muscles of the human body
- Muscle atrophy
- Muscle memory
- Muscle tone (residual muscle tension)
- Musculoskeletal system
- Myopathy (pathology of muscle cells)
- Myotomy
- Phonomyography
- Preflexes
- Rapid plant movement
- Rohmert's law

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External links

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- University of Dundee article on performing neurological examinations (Quadriceps "strongest")
- Muscle efficiency in rowing
- Human Muscle Tutorial (clear pictures of main human muscles and their Latin names, good for orientation)
- Microscopic stains of skeletal and cardiac muscular fibers to show striations. Note the differences in myofibrilar arrangements.

Retrieved from "http://en.wikipedia.org/wiki/Muscle"
Categories: Muscular system | Tissues | Exercise physiology

List of neurological disorders

From Wikipedia, the free encyclopedia

This is a list of major and frequently observed neurological disorders (e.g., Alzheimer's disease), symptoms (e.g., back pain), signs (e.g., aphasia) and syndromes (e.g., Aicardi syndrome).

Contents: Top - 0–9 A B C D E F G H I J K L M N O P Q R S T U V W X Y Z

A

- Abarognosis
- Acquired Epileptiform Aphasia
- Acute disseminated encephalomyelitis
- Adrenoleukodystrophy
- Agenesis of the corpus callosum
- Agnosia
- Aicardi syndrome
- Alexander disease
- Alpers' disease
- Alternating hemiplegia
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- Amyotrophic lateral sclerosis (see Motor Neurone Disease)
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- Angelman syndrome
- Angiomatosis
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U

V

Y

MERCK

Vascular Disorders

Vascular disorders that affect vision include central and branch retinal artery or vein occlusion, ischemic optic neuropathy, amaurosis fugax, occipital lobe stroke, and temporal arteritis (which occurs primarily in the elderly).

Atherosclerotic cardiovascular risk factors underlie almost all ophthalmic vascular disorders, and treatment focuses on management of these risk factors. The leading cause of death in patients with an ophthalmic vascular disorder is a cardiovascular disorder.

Retinal Artery Occlusion

Retinal artery occlusion causes painless, sudden, unilateral blindness.

The most common cause of retinal artery occlusion in the elderly is embolization of a thrombus or an atheroma from the carotid artery to the central retinal artery in the optic nerve head. Less common causes include temporal arteritis, optic neuritis, hypercoagulability, and, rarely, severely elevated intraocular pressure (IOP). Within an hour of occlusion, reactive arterial spasm ceases, and some blood flow is restored to the retina, which then appears relatively normal through an ophthalmoscope. However, within several hours the retina becomes edematous and gray because ischemia continues and retinal ganglion cells die. Because the retina in the foveal area contains no ganglion cells, the reddish underlying choroid remains visible, accounting for the characteristic, central, cherry-red spot surrounded by the gray retina. In 2 to 3 wk, the cherry-red spot disappears, and as the ganglion cells and their axons die, the optic nerve becomes white--the hallmark of primary optic atrophy.

A retinal artery branch may become occluded when an atheroma breaks off and passes through the central retinal artery. The occlusion (called a Hollenhorst plaque) can usually be seen as a refractile object in the branch. This finding indicates embolic activity, usually originating in the carotid system. The portion of the retina supplied by the occluded vessel stops functioning, resulting in a visual field defect that may not affect central vision.

Intervention is rarely possible because it is needed within 90 min of the occlusion to prevent retinal cell death. Rapidly reducing IOP by paracentesis plus vasodilators occasionally induces the embolus to move more peripherally, limiting vision loss in the affected area. Other treatments (eg, eyeball massage to improve O_2 delivery to tissues, CO_2 therapy to promote vasodilation, oral anticoagulants, thrombolytics) may be attempted. None has proved effective, and thrombolytics may have serious adverse effects. Patients should also be evaluated for atherosclerotic risk factors, which should be managed. Anticoagulants may help decrease risk of future emboli.

Retinal Vein Occlusion

Retinal vein occlusion causes painless, sudden, usually unilateral blindness.

Retinal vein occlusion (RVO) is probably the most common ophthalmic vascular disorder and occurs most commonly among people with atherosclerosis or glaucoma. Less common causes include leukemia and lymphoma, autoimmune disorders, and hypercoagulability disorders. RVO is classified as nonischemic or ischemic and may affect the central retinal vein or a branch.

Central RVO: Symptoms are similar to those of central retinal artery occlusion--sudden, painless, typically severe, unilateral vision loss. After central RVO occurs, some minimal vision may remain. About 10% of patients who develop central RVO in one eye later develop central RVO in the other eye.

Diagnosis is by ophthalmoscopy. Findings include distended, tortuous veins with massive hemorrhages and edema throughout the retina. The margins of the optic nerve become blurred, and the disk becomes swollen. Complete resorption of the hemorrhages and edema may take months or even years.

Fluorescein angiography helps differentiate nonischemic from ischemic forms. Ischemic RVO is characterized by relatively large retinal areas of capillary nonperfusion (which may require retinal laser photocoagulation if neovascularization occurs).

Prognosis is poor for elderly patients. About 25% develop a fibrovascular membrane that seals the aqueous humor outflow channels in the anterior chamber, resulting in a painful, secondary neovascular glaucoma in 3 to 6 mo; without treatment, blindness occurs within weeks. Patients with the nonischemic form have a better visual prognosis than those with the ischemic form.

Central RVO is most often treated with retinal laser photocoagulation, but its effectiveness is still being assessed. Systemic anticoagulation is not typically recommended. Intravitreal injection of triamcinolone acetonide may help decrease macular edema and improve visual acuity in some patients with central RVO.

Branch RVO: This disorder is similar to central RVO, but a branch of the central retinal vein is obstructed, most often the superior temporal branch. Vision is usually unaffected unless the retinal swelling impinges on the macula. Visual field defects, if present, depend on which retinal quadrant is involved. Neovascular glaucoma develops much less often in branch RVO than in central RVO.

Diagnosis is similar to that for central RVO. Ophthalmoscopic findings include exudates and hemorrhages confined to the involved retinal quadrant. Laser photocoagulation helps preserve vision.

Ischemic Optic Neuropathy

Ischemic optic neuropathy is inadequate blood supply to the optic nerve, sometimes causing blindness.

Ischemic optic neuropathy (ION) usually occurs only in people > 60. Most cases are nonarteritic and attributed to the effects of atherosclerosis, diabetes, or hypertension on optic nerve perfusion. Temporal arteritis causes about 5% of cases (arteritic ION).

Symptoms and signs are sudden, partial or complete vision loss, accompanied by swelling of the optic nerve head and often hemorrhage. Visual field defects may manifest as loss of half the visual field with a horizontal demarcation or as central or centrocecal (surrounding the natural blind spot) scotomata. Decreased vision is soon followed by pallor of the optic disk. When temporal arteritis is the cause, tenderness along the temporal artery may be noted, as well as headache, jaw pain while chewing, fever, malaise, anorexia, weight loss, and joint and muscle pain.

Diagnosis of nonarteritic ION is presumptive based on symptoms, signs, and presence of

atherosclerotic risk factors. Diagnosis of arteritic ION is suggested by symptoms and signs and supported by a dramatically elevated Westergren ESR (normal: <= [age + 10]/2 for women and age/2 for men), an elevated C-reactive protein level, or both. Diagnosis is confirmed by temporal artery biopsy showing granulomatous inflammatory changes.

For nonarteritic ION, treatment does not help, but atherosclerotic risk factors should be managed. Most patients have at least some return of vision. Vision loss in the other eye may occur months or years later.

For arteritic ION, treatment is IV methylprednisolone (1 g/day for the first 3 to 5 days), after which oral prednisone (60 mg/day) can be used and tapered slowly over 3 to 12 mo or more, depending on response. Corticosteroids should be started immediately to protect the other eye; treatment should not be postponed for confirmation by biopsy. Long-term anticoagulant therapy may help selected elderly patients with a history of amaurosis fugax suggesting atheromatosis.

Amaurosis Fugax

Amaurosis fugax is acute vision loss lasting minutes to hours. It usually involves only part of a visual field of one eye.

Amaurosis fugax is a symptom that suggests retinal or optic nerve ischemia caused by atherosclerosis or an embolus in a carotid or thoracic aortic artery; this symptom occasionally indicates migraine headache. Patients > 50 are most susceptible. Risk factors are those for atherosclerosis and a family history of stroke.

Amaurosis fugax manifests as a dimming of vision in one eye, sometimes perceived as a window shade being partially or completely drawn over the eye. Recovery of clear vision begins within 5 to 10 min and occurs in the reverse order from the onset pattern. Several episodes may precede an attack of ischemic optic neuropathy or stroke. The annual risk of stroke after amaurosis fugax is about 2%. Amaurosis fugax can be bilateral if associated with low BP.

If amaurosis fugax is accompanied by hemiplegia on the side opposite the affected eye (indicating a transient ischemic attack), carotid stenosis on the side of the affected eye should be strongly suspected. Early recognition of severe carotid stenosis is important because without appropriate medical (eg, daily aspirin) and surgical (eg, carotid endarterectomy) intervention, permanent vision loss or hemiplegia often results. Aortic arch syndrome may be suspected if blackouts become increasingly frequent and are related to changes in posture (eg, suddenly sitting up or standing).

Occipital Lobe Stroke

Occipital lobe stroke is caused by a vascular lesion in the vertebral-basilar system and causes sudden, sometimes total blindness.

Infarction in one or both occipital lobes may result from local atheromas or emboli in the vertebral-basilar system. An occipital lobe stroke, usually resulting from posterior cerebral artery infarction, is characterized by sudden onset of congruous homonymous hemianopia. Total blindness occurs suddenly; however, within minutes, some vision returns in the ipsilateral homonymous visual field. Bilateral posterior occlusions usually occur simultaneously. Thrombosis of the basilar artery also causes bilateral homonymous hemianopia.

As with any ischemic stroke, treatment with aspirin or other anticoagulants is indicated. Some vision returns in almost all patients with cortical blindness.

This topic was last updated May 2006.

Symptoms

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PAIN

Pain is the chief symptom of most musculoskeletal disorders. The pain may be mild or severe, local or widespread (diffuse). Although pain may be acute and short-lived, as is the case with most injuries, pain may be ongoing with chronic illnesses, such as rheumatoid arthritis.

Causes

Musculoskeletal pain can be caused by damage to bones, joints, muscles, tendons, ligaments, bursae, or nerves. Injuries are the most common cause. If no injury has occurred or if pain persists for more than a few days, then another cause is often responsible.

Bone pain is usually deep, penetrating, or dull. It commonly results from injury. Other less common causes of bone pain include bone infection (osteomyelitis) and tumors.

Muscle pain is often less intense than that of bone pain but can be very unpleasant. For example, a muscle spasm or cramp (a sustained painful muscle contraction) in the calf is an intense pain that is commonly called a charleyhorse. Pain can occur when a muscle is affected by an injury, an autoimmune reaction (for example, polymyositis or dermatomyositis), loss of blood flow to the muscle, infection, or invasion by a tumor.

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Pronunciations

acetaminophen
amyotrophic lateral sclerosis
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arteritis

Tendon and ligament pain is often less intense than bone pain. It is often worse when the affected tendon or ligament is stretched or moved. Common causes of tendon pain include tendinitis, tenosynovitis, lateral and medial epicondylitis, and tendon injuries. Common causes of ligament pain include injuries (sprains).

Fibromyalgia may cause pain in the muscles, tendons, or ligaments. The pain is usually in multiple locations and may be difficult to describe precisely. Affected people usually have other symptoms.

Virtually all joint injuries and diseases produce a stiff, aching pain, often referred to as "arthritic" pain. The pain is worse when the joint is moved and may range from mild to severe. With some conditions, there may be swelling of the joint along with the pain. Joint inflammation (arthritis) is a common cause of joint pain. There are many types of arthritis, including rheumatoid and other types of inflammatory arthritis, osteoarthritis, infectious arthritis, and arthritis due to gout or pseudogout. Other causes of joint pain include autoimmune and vasculitic disorders (for example, systemic lupus erythematosus, polymyalgia rheumatica, and polyarteritis nodosa), avascular necrosis of bone, and injuries (for example, dislocations, sprains, and fractures affecting the portion of the bone inside the

joint). Sometimes, pain originating in structures near the joint, such as tendons and bursae, seems to be coming from the joint.

Some musculoskeletal disorders cause pain by compressing nerves. These conditions include the "tunnel syndromes" (for example, carpal tunnel syndrome, cubital tunnel syndrome, and tarsal tunnel syndrome). The pain tends to radiate along the path supplied by the nerve and may be burning.

Bursal pain can be caused by bursitis or fibromyalgia. Usually, bursal pain is worse with movement involving the bursa. There may be swelling.

Sometimes, pain that seems to be musculoskeletal is actually caused by a disorder in another organ system. For instance, shoulder pain may be caused by a disorder affecting the spleen or gallbladder. Back pain may be caused by an abdominal aortic aneurysm. Arm pain may be caused by a heart attack (myocardial infarction). Additionally, sometimes pain that seems to be coming from one part of the musculoskeletal system actually comes from another part. For instance, knee pain in an adolescent may be caused by a disorder of the hip called slipped capital femoral epiphysis.

Evaluation and Treatment

Sometimes, the type of pain suggests where the pain has originated. For example, pain that worsens with motion suggests a musculoskeletal disorder. Pain with muscle spasm suggests that pain is caused by a muscle disorder. The site of swelling or the location of tenderness when the doctor palpates the area (for example, a joint, ligament, or bursa) often indicates the source of pain. However, often these characteristics of pain do not indicate its origin or cause. Thus, doctors usually base a specific diagnosis on the presence of other symptoms and often on the results of laboratory tests and x-rays. For example, Lyme disease often produces joint pain and a bull's eye—like skin rash; blood tests show antibodies to the bacteria that cause Lyme disease. Gout is characterized by a sudden attack of pain, swelling, and redness in the joint at the base of the big toe or other joints; tests of the joint fluid generally show the presence of uric acid crystals.

Blood tests are useful only in supporting the diagnosis made by the doctor after an examination. A diagnosis is not made or confirmed by a blood test alone. Examples of such blood tests include rheumatoid factor and antinuclear antibodies, which are used to help diagnose many of the common causes of arthritis, such as rheumatoid arthritis and systemic lupus erythematosus. Usually, such tests are recommended only if symptoms specifically suggest such a disorder or are persistent or unusually severe.

X-rays are primarily used to take images of bones; they do not show muscles, tendons and ligaments. X-rays are usually taken if the doctor suspects a fracture or, less commonly, a bone tumor or infection or to look for changes that confirm a person has a certain kind of arthritis (for example, rheumatoid arthritis or osteoarthritis).

A computed tomography (CT) scan is more sensitive than an x-ray and is often used to obtain more detail about a fracture or bone problem that was found with plain x-rays.

Unlike plain x-rays, magnetic resonance imaging (MRI) can identify abnormalities of soft tissues such as muscles, bursae, ligaments, and tendons. Thus, MRI may be used when the doctor suspects damage to a major ligament or tendon, or damage to important structures inside a joint.

Pain is usually best relieved by treating its cause. In addition, the doctor may recommend analgesics (see Pain: Treatment) such as acetaminophen , nonsteroidal anti-inflammatory drugs (NSAIDs), or, if pain is severe, opioids. Depending on the cause, applying cold or heat or immobilizing the joint may help relieve musculoskeletal pain.

DIFFICULTY MOVING

A person may have difficulty moving all or part of the body.

Causes

Moving may be difficult because of disorders that restrict joint motion or that produce weakness. Movement may also be limited when motion causes pain. Certain nervous system abnormalities interfere with movement without causing pain or weakness. For example, Parkinson's disease causes muscle stiffness, tremor, and difficulty initiating movement.

Joint Disorders: A joint that is stiffened by scar tissue from a previous injury can have limited range and speed of motion. When a normal joint is not used, it may stiffen. For example when a person's arm is paralyzed by a stroke or even placed in a sling for a period of time, the joints in the shoulder and elbow may develop scar tissue that freezes the joint in place if the arm is not regularly flexed and stretched. Fluid that accumulates in a joint from arthritis or an acute injury can interfere with joint motion. A piece of torn cartilage from an injury (typically in the knee) may block joint motion.

Weakness: Although many people complain of weakness when they feel tired or run down, true weakness means that full effort does not generate normal muscle contractions. Normal voluntary muscle contraction requires that the brain generate a signal that then travels through the spinal cord and nerves to reach a normally functioning muscle. Therefore, true weakness can result from injury or disease affecting the nervous system, muscles, or connections between them (neuromuscular junction).

Brain problems include strokes, injuries, tumors, and degenerative disorders (such as multiple sclerosis, which also can affect the spinal cord and nerves). Spinal cord disorders include injury, bleeding, and tumors. Spinal nerve roots can be affected by a ruptured intervertebral disk, and peripheral nerves by injury or polyneuropathy. The neuromuscular junction can be affected by myasthenia gravis, drugs such as botulinum toxin injections, and certain poisons such as organophosphates (used in nerve gas and many insecticides).

Muscle disorders causing weakness include muscular dystrophy and polymyositis. The muscle weakness that commonly occurs following immobilization (in a cast or from prolonged bed rest) and in old age is due to a reduction in muscle mass (sarcopenia) and results from lack of use. The remaining muscle mass functions normally, but there is not an adequate amount.

Weakness may be limited to one extremity or part of an extremity, as is typically the case when a single nerve, joint, or muscle is affected, or diffuse, as occurs in widespread neurologic or muscular diseases.

Pain: People with pain in the muscles, ligaments, bones, or joints tend to consciously and unconsciously limit motion. This often gives the impression of weakness even though the nervous system and muscles are able to generate movement.

Evaluation and Treatment

Doctors can often diagnose weakness based on the person's symptoms and the results of the physical examination. Doctors first try to determine whether the person can contract the muscles normally. If the person can contract the muscles normally but has trouble moving a joint, the doctor tries to move the joint for the person while the person relaxes (passive motion). If motion is painful, inflammation may be the problem. If passive motion causes little pain but is blocked, joint contracture (for example, due to scar tissue) may be the problem.

If passive motion is neither painful nor blocked, the person is giving full effort, and there is no sign of Parkinson's disease or other neurologic disorder causing difficulty initiating movement, then true muscle weakness is likely. The cause of true muscle weakness can often be determined by noting the person's symptoms, which muscles are affected, whether muscles have shrunk, and muscle tone and by testing the person's reflexes with a reflex hammer. For example, if weakness affects mainly the large muscles such as the hips,

thighs, and shoulders, the cause may be a disorder producing widespread damage to muscles. If weakness affects mainly the eye muscles (causing double vision), the cause may be a disorder of the neuromuscular junction. If weakness affects mainly the fingers, hands, and feet, particularly if there is loss of sensation, the cause may be a disorder that damages many nerves (polyneuropathy). The nerves to the fingers, hands, and feet are the body's longest and thus the most vulnerable peripheral nerves. If muscles have shrunk, the disorder causing the problem has been present for months or years. If the person's reflexes are decreased or slow, the cause may be nerve damage. If reflexes are increased or more rapid than expected, the cause may be spinal cord or brain damage. The doctor checks muscle tone by testing passive movement. Muscle tone may be decreased when weakness results from a peripheral nerve disorder. Muscle tone may be increased when weakness results from a spinal cord or brain disorder.

If the cause is still not clear, other tests can help. Disorders of the brain or spinal cord are diagnosed using neuroimaging tests such as CT or MRI. To differentiate between weakness caused by damage to the peripheral nerves, muscles, and neuromuscular junction, tests such as electromyography and nerve conduction velocity (see Diagnosis of Brain, Spinal Cord, and Nerve Disorders: Electromyography and Nerve Conduction Studies) usually help. Certain other disorders (for example, low blood levels of potassium or vitamin D) are diagnosed with blood tests.

For joints that are fixed, joint flexibility can be maximized by stretching exercises and physical therapy. If the joint's range of motion is severely restricted by scar tissue, surgery may be necessary. The only way to relieve weakness is to treat the disorder causing it.

Classifying Weakness

Underlying Problem	Example	Description
Muscle disease	Muscular dystrophies	A group of inherited muscle disorders that leads to muscle weakness of varying severity
	Infections or inflammatory disorders (acute viral myositis, polymyositis)	Muscles tender or painful and weak
Widespread muscle damage caused by use of a drug (drug-induced myopathy)	Myopathy due to corticosteroids, statins, lithium, alcohol, clofibrate, colchicine	Weakness usually begins at the hips and may spread to other muscles; pain may be absent
Low blood levels of potassium	Hypokalemic myopathy (caused by certain disorders or use of diuretics)	The person experiences periods of weakness throughout the body
Abnormal levels of thyroid hormone	High levels of thyroid hormone (hyperthyroidism) or low levels of thyroid hormone (hypothyroidism)	High or low levels of thyroid hormone produce weakness that is usually more pronounced in the shoulders and hips than in the hands and feet
Low levels of vitamin D	Osteomalacia	Pain in the back, with weakness in the legs; rarely pain throughout the body
Disease of the neuromuscular junction	Myasthenia gravis, curare toxicity, Eaton-Lambert syndrome, insecticide poisoning, botulism, diphtheria	Weakness or paralysis affecting all or many muscles; sometimes affects mainly eye muscles
Damage to a single nerve (mononeuropathy)	Diabetic neuropathy, local pressure	Weakness or paralysis of muscles and loss of sensation in the area served by the injured nerve
Damage to many nerves (polyneuropathy)	Diabetes, Guillain-Barre syndrome, folate deficiency, toxins, drugs	Weakness or paralysis of muscles and loss of sensation in the areas served by the affected nerves

Spinal nerve root damage	Ruptured disk in the spine of the neck or lower back	Pain in the neck and weakness or numbness in an arm, low back pain shooting down the leg (sciatica), and leg weakness or numbness
Degeneration of nerve cell bodies in the spinal cord	Amyotrophic lateral sclerosis	Progressive loss of muscle bulk and strength, but no loss of sensation
Spinal cord damage	Trauma to the neck or back, spinal cord tumors, spinal stenosis, multiple sclerosis, transverse myelitis, vitamin B_{12} deficiency	Weakness or paralysis of the arms and legs below the level of injury, progressive loss of sensation below the level of injury, back pain; bowel, bladder, and sexual function are affected
Brain damage	Strokes, tumors, head trauma, multiple sclerosis, infections	Weakness or paralysis of muscles in the area served by the injured part of the brain, often with other symptoms of brain damage
Psychological problems	Depression, imagined symptoms or hysteria (conversion reaction)	Complaint of whole-body weakness or paralysis with no evidence of nerve damage

JOINT STIFFNESS

Stiffness is the feeling that motion of a joint is limited or difficult. The feeling is not caused by weakness or reluctance to move the joint due to pain. Some people with stiffness are capable of moving the joint through its full range of motion. Joint stiffness usually occurs or is worse immediately after awakening or resting. Stiffness is common with arthritis. Morning stiffness commonly occurs with rheumatoid arthritis and other types of inflammatory arthritis in which stiffness typically occurs on arising and gradually lessens with activity only after an hour or two.

Doctors can sometimes diagnose the cause of stiffness by the person's symptoms and the results of a physical examination. The person is examined to make sure that the problem is not pain with motion or weakness. Because arthritis is often the cause, blood tests (for example, rheumatoid factor and antinuclear antibodies) and x-rays may be done.

Stiffness is relieved by treating the disorder causing it. Stretching, physical therapy, and taking a hot shower on arising may improve the ability to perform activities that require flexibility.

JOINT NOISES

Joint noises, such as creaks and clicks, are common in many people, but they can also occur with specific problems of the joints. For example, the base of the knee cap may creak when it is damaged by osteoarthritis, and the jaw may click in a person who has temporomandibular joint disorder. Doctors ask about the person's symptoms and perform an examination to determine whether a joint noise is a symptom of a certain disorder. Further evaluation and treatment are needed only if the evaluation suggests a significant joint problem. Joint noises themselves do not require treatment.

Last full review/revision September 2006 by Michael Jacewicz, MD

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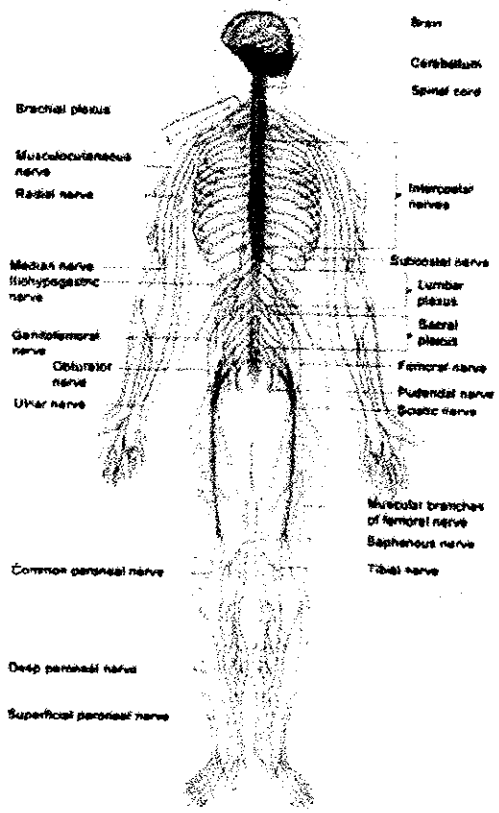
Peripheral nervous system

From Wikipedia, the free encyclopedia

The **peripheral nervous system (PNS)** resides or extends outside the central nervous system (CNS), which consists of the brain and spinal cord.[1] The main function of the PNS is to connect the CNS to the limbs and organs. Unlike the central nervous system, the PNS is not protected by bone or by the blood-brain barrier, leaving it exposed to toxins and mechanical injuries. The peripheral nervous system is divided into the somatic nervous system and the autonomic nervous system; some textbooks also include sensory systems.[2]

Brain: Peripheral nervous system



The Human Nervous System. Blue is PNS while red is CNS.

Latin *Pars peripherica; Systema nervosum periphericum*

Contents

General classification

By direction

There are two types of directions of the neurons: sensory neurons are afferent (i.e. relaying impulses TO the central nervous system), motor neurons are efferent (i.e. relaying impulses FROM the central nervous system). However, there are relay neurons in the CNS as well .

By function

The peripheral nervous system is functionally as well as structurally divided into the somatic nervous system and autonomic nervous system. The somatic nervous system is responsible for coordinating the body movements, and also for receiving external stimuli. It is the system that regulates activities that are under conscious control. The autonomic nervous system is then split into the sympathetic division, parasympathetic division, and enteric division. The *sympathetic nervous system* responds to impending danger or stress, and is responsible for the increase of one's heartbeat and blood pressure, among other

Headache

From Wikipedia, the free encyclopedia

In medicine a **headache** or **cephalalgia** is a symptom of a number of different conditions of the head[1]. Some of the causes are benign while others are medical emergencies.

There are a number of different classification systems for headaches. The most well-recognized is that of the International Headache Society.

Treatment of a headache depends on the underlying etiology or cause, but commonly involves analgesics.

Headache

ICD-10	G43.-G44., R51.
ICD-9	339, 784.0
DiseasesDB	19825
MedlinePlus	003024
eMedicine	neuro/517 neuro/70
MeSH	D006261

Contents

Classification

The first recorded classification system that resembles the modern ones was published by Thomas Willis, in *De Cephalagia* in 1672. In 1787 Christian Baur generally divided headaches into idiopathic (primary headaches) and symptomatic (secondary ones), and defined 84 categories.[2]

Today headaches are most thoroughly classified by the International Headache Society's International Classification of Headache Disorders (ICHD), which published the second edition in 2004.[3] This classification is accepted by the WHO.[4]

Other classification systems exist. One of the first published attempts was in 1951.[5] The National Institutes of Health developed a classification system in 1962.

Headaches can also be classified by severity and acuity of onset. Headaches that are both severe and acute are known as thunderclap headaches.

Before forming three cords

The first nerve off the brachial plexus, or plexus brachialis, is the dorsal scapular nerve, arising from C5 nerve root, and innervating the rhomboids and the levator scapulae muscles. The long thoracic nerve arises from C5, C6 and C7 to innervate the serratus anterior. The brachial plexus first forms three trunks, the superior trunk, composed of the C5 and C6 nerve roots, the middle trunk, made of the C7 nerve root, and the inferior trunk, made of the C8 and T1 nerve roots. The suprascapular nerve is an early branch of the superior trunk. It innervates the suprascapular and infrascapular muscles, part of the rotator cuff. The trunks reshuffle as they traverse towards the arm into cords. There are three of them. The lateral cord is made up of fibers from the superior and middle trunk. The posterior cord is made up of fibers from all three trunks. The medial cord is composed of fibers solely from the inferior trunk.

Lateral cord

The lateral cord gives rise to the following nerves:

- The lateral pectoral nerve, C5, C6 and C7 to the pectoralis major muscle, or musculus pectoralis major.
- The musculocutaneous nerve which innervates the biceps muscle
- The median nerve, partly. The other part comes from the medial cord. See below for details.

Posterior cord

The posterior cord gives rise to the following nerves:

- The upper subscapular nerve, C7 and C8, to the subscapularis muscle, or musculus supca of the rotator cuff.
- The lower subscapular nerve, C5 and C6, to the teres major muscle, or the musculus teres major.
- The thoracodorsal nerve, C6, C7 and C8, to the latissimus dorsi muscle, or musculus latissimus dorsi.
- The axillary nerve, which supplies sensation to the shoulder and motor to the deltoid muscle or musculus deltoideus, and the teres minor muscle, or musculus teres minor, also of the rotator cuff.
- The radial nerve, or nervus radialis, which innervates the triceps brachii muscle, the brachioradialis muscle, or musculus brachioradialis,, the extensor muscles of the fingers and wrist (extensor carpi radialis muscle), and the extensor and abductor muscles of the thumb. See radial nerve injuries.

Medial cord

The medial cord gives rise to the following nerves:

- The median pectoral nerve, C8 and T1, to the pectoralis muscle
- The medial brachial cutaneous nerve, T1
- The medial antebrachial cutaneous nerve, C8 and T1
- The median nerve, partly. The other part comes from the lateral cord. C7, C8 and T1 nerve roots. The first branch of the median nerve is to the pronator teres muscle, then the flexor carpi radialis, the palmaris longus and the flexor digitorum superficialis. The median nerve provides sensation to the anterior palm, the anterior thumb, index finger and middle finger. It is the nerve compressed in carpal tunnel syndrome.
- The ulnar nerve originates in nerve roots C7, C8 and T1. It provides sensation to the ring and

physiological changes, along with the sense of excitement one feels due to the increase of adrenaline in the system. The *parasympathetic nervous system*, on the other hand, is evident when a person is resting and feels relaxed, and is responsible for such things as the constriction of the pupil, the slowing of the heart, the dilation of the blood vessels, and the stimulation of the digestive and genitourinary systems. The role of the *enteric nervous system* is to manage every aspect of digestion, from the esophagus to the stomach, small intestine and colon.

Naming of specific nerves

Ten out of the twelve cranial nerves originate from the brainstem, and mainly control the functions of the anatomic structures of the head with some exceptions. The nuclei of cranial nerves I and II lie in the forebrain and thalamus, respectively, and are thus not considered to be true cranial nerves. CN X (10) receives visceral sensory information from the thorax and abdomen, and CN XI (11) is responsible for innervating the sternocleidomastoid and trapezius muscles, neither of which is exclusively in the head.

Spinal nerves take their origins from the spinal cord. They control the functions of the rest of the body. In humans, there are 31 pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral and 1 coccygeal. In the cervical region, the spinal nerve roots come out *above* the corresponding vertebrae (i.e. nerve root between the skull and 1st cervical vertebrae is called spinal nerve C1). From the thoracic region to the coccygeal region, the spinal nerve roots come out *below* the corresponding vertebrae. It is important to note that this method creates a problem when naming the spinal nerve root between C7 and T1 (so it is called spinal nerve root C8). In the lumbar and sacral region, the spinal nerve roots travel within the dural sac and they travel below the level of L2 as the cauda equina.

Cervical spinal nerves (C1-C4)

Further information: Cervical plexus

The first 4 cervical spinal nerves, C1 through C4, split and recombine to produce a variety of nerves that subserve the neck and back of head.

Spinal nerve C1 is called the suboccipital nerve which provides motor innervation to muscles at the base of the skull. C2 and C3 form many of the nerves of the neck, providing both sensory and motor control. These include the greater occipital nerve which provides sensation to the back of the head, the lesser occipital nerve which provides sensation to the area behind the ears, the greater auricular nerve and the lesser auricular nerve. See occipital neuralgia. The phrenic nerve arises from nerve roots C3, C4 and C5. It innervates the diaphragm, enabling breathing. If the spinal cord is transected above C3, then spontaneous breathing is not possible. See myelopathy

Brachial plexus (C5-T1)

Further information: Brachial plexus

The last four cervical spinal nerves, C5 through C8, and the first thoracic spinal nerve, T1,combine to form the brachial plexus, or plexus brachialis, a tangled array of nerves, splitting, combining and recombining, to form the nerves that subserve the arm and upper back. Although the brachial plexus may appear tangled, it is highly organized and predictable, with little variation between people. See brachial plexus injuries.

pinky fingers. It innervates the flexor carpi ulnaris muscle, the flexor digitorum profundus muscle to the ring and pinky fingers, and the intrinsic muscles of the hand (the interosseous muscle, the lumbrical muscles and the flexor pollicus brevis muscle). This nerve traverses a groove on the elbow called the cubital tunnel, also known as the funny bone. Striking the nerve at this point produces an unpleasant sensation in the ring and little finger.

Neurotransmitters

The main neurotransmitters of the peripheral nervous system are acetylcholine and noradrenaline. However, there are several other neurotransmitters as well, jointly labeled Non-noradrenergic, non-cholinergic (NANC) transmitters. Examples of such transmitters include non-peptides: ATP, GABA, dopamine, NO, and peptides: neuropeptide Y, VIP, GnRH, Substance P and CGRP. [3]

References

1. ^ *peripheral nervous system* at Dorland's Medical Dictionary
2. ^ Maton, Anthea; Jean Hopkins, Charles William McLaughlin, Susan Johnson, Maryanna Quon Warner, David LaHart, Jill D. Wright (1993). *Human Biology and Health*. Englewood Cliffs, New Jersey, USA: Prentice Hall. pp. 132–144. ISBN 0-13-981176-1.
3. ^ Pharmacology, (Rang, Dale, Ritter & Moore, ISBN 0443071454, 5:th ed., Churchill Livingstone 2003). Page 132.

Z

Symptoms

PAIN

Pain is the chief symptom of most musculoskeletal disorders. The pain may be mild or severe, local or widespread (diffuse). Although pain may be acute and short-lived, as is the case with most injuries, pain may be ongoing with chronic illnesses, such as rheumatoid arthritis.

Causes

Musculoskeletal pain can be caused by damage to bones, joints, muscles, tendons, ligaments, bursae, or nerves. Injuries are the most common cause. If no injury has occurred or if pain persists for more than a few days, then another cause is often responsible.

Bone pain is usually deep, penetrating, or dull. It commonly results from injury. Other less common causes of bone pain include bone infection (osteomyelitis) and tumors.

Muscle pain is often less intense than that of bone pain but can be very unpleasant. For example, a muscle spasm or cramp (a sustained painful muscle contraction) in the calf is an intense pain that is commonly called a charleyhorse. Pain can occur when a muscle is affected by an injury, an autoimmune reaction (for example, polymyositis or dermatomyositis), loss of blood flow to the muscle, infection, or invasion by a tumor.

Pronunciations

acetaminophen
amyotrophic lateral sclerosis
aneurysm
aortic aneurysm
arteritis

Tendon and ligament pain is often less intense than bone pain. It is often worse when the affected tendon or ligament is stretched or moved. Common causes of tendon pain include tendinitis, tenosynovitis, lateral and medial epicondylitis, and tendon injuries. Common causes of ligament pain include injuries (sprains).

Fibromyalgia may cause pain in the muscles, tendons, or ligaments. The pain is usually in multiple locations and may be difficult to describe precisely. Affected people usually have other symptoms.

Virtually all joint injuries and diseases produce a stiff, aching pain, often referred to as "arthritic" pain. The pain is worse when the joint is moved and may range from mild to severe. With some conditions, there may be swelling of the joint along with the pain. Joint inflammation (arthritis) is a common cause of joint pain. There are many types of arthritis, including rheumatoid and other types of inflammatory arthritis, osteoarthritis, infectious arthritis, and arthritis due to gout or pseudogout. Other causes of joint pain include autoimmune and vasculitic disorders (for example, systemic lupus erythematosus, polymyalgia rheumatica, and polyarteritis nodosa), avascular necrosis of bone, and injuries (for example, dislocations, sprains, and fractures affecting the portion of the bone inside the

joint). Sometimes, pain originating in structures near the joint, such as tendons and bursae, seems to be coming from the joint.

Some musculoskeletal disorders cause pain by compressing nerves. These conditions include the "tunnel syndromes" (for example, carpal tunnel syndrome, cubital tunnel syndrome, and tarsal tunnel syndrome). The pain tends to radiate along the path supplied by the nerve and may be burning.

Bursal pain can be caused by bursitis or fibromyalgia. Usually, bursal pain is worse with movement involving the bursa. There may be swelling.

Sometimes, pain that seems to be musculoskeletal is actually caused by a disorder in another organ system. For instance, shoulder pain may be caused by a disorder affecting the spleen or gallbladder. Back pain may be caused by an abdominal aortic aneurysm. Arm pain may be caused by a heart attack (myocardial infarction). Additionally, sometimes pain that seems to be coming from one part of the musculoskeletal system actually comes from another part. For instance, knee pain in an adolescent may be caused by a disorder of the hip called slipped capital femoral epiphysis.

Evaluation and Treatment

Sometimes, the type of pain suggests where the pain has originated. For example, pain that worsens with motion suggests a musculoskeletal disorder. Pain with muscle spasm suggests that pain is caused by a muscle disorder. The site of swelling or the location of tenderness when the doctor palpates the area (for example, a joint, ligament, or bursa) often indicates the source of pain. However, often these characteristics of pain do not indicate its origin or cause. Thus, doctors usually base a specific diagnosis on the presence of other symptoms and often on the results of laboratory tests and x-rays. For example, Lyme disease often produces joint pain and a bull's eye—like skin rash; blood tests show antibodies to the bacteria that cause Lyme disease. Gout is characterized by a sudden attack of pain, swelling, and redness in the joint at the base of the big toe or other joints; tests of the joint fluid generally show the presence of uric acid crystals.

Blood tests are useful only in supporting the diagnosis made by the doctor after an examination. A diagnosis is not made or confirmed by a blood test alone. Examples of such blood tests include rheumatoid factor and antinuclear antibodies, which are used to help diagnose many of the common causes of arthritis, such as rheumatoid arthritis and systemic lupus erythematosus. Usually, such tests are recommended only if symptoms specifically suggest such a disorder or are persistent or unusually severe.

X-rays are primarily used to take images of bones; they do not show muscles, tendons and ligaments. X-rays are usually taken if the doctor suspects a fracture or, less commonly, a bone tumor or infection or to look for changes that confirm a person has a certain kind of arthritis (for example, rheumatoid arthritis or osteoarthritis).

A computed tomography (CT) scan is more sensitive than an x-ray and is often used to obtain more detail about a fracture or bone problem that was found with plain x-rays.

Unlike plain x-rays, magnetic resonance imaging (MRI) can identify abnormalities of soft tissues such as muscles, bursae, ligaments, and tendons. Thus, MRI may be used when the doctor suspects damage to a major ligament or tendon, or damage to important structures inside a joint.

Pain is usually best relieved by treating its cause. In addition, the doctor may recommend analgesics (see Pain: Treatment) such as acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), or, if pain is severe, opioids. Depending on the cause, applying cold or heat or immobilizing the joint may help relieve musculoskeletal pain.

DIFFICULTY MOVING

A person may have difficulty moving all or part of the body.

Causes

Moving may be difficult because of disorders that restrict joint motion or that produce weakness. Movement may also be limited when motion causes pain. Certain nervous system abnormalities interfere with movement without causing pain or weakness. For example, Parkinson's disease causes muscle stiffness, tremor, and difficulty initiating movement.

Joint Disorders: A joint that is stiffened by scar tissue from a previous injury can have limited range and speed of motion. When a normal joint is not used, it may stiffen. For example when a person's arm is paralyzed by a stroke or even placed in a sling for a period of time, the joints in the shoulder and elbow may develop scar tissue that freezes the joint in place if the arm is not regularly flexed and stretched. Fluid that accumulates in a joint from arthritis or an acute injury can interfere with joint motion. A piece of torn cartilage from an injury (typically in the knee) may block joint motion.

Weakness: Although many people complain of weakness when they feel tired or run down, true weakness means that full effort does not generate normal muscle contractions. Normal voluntary muscle contraction requires that the brain generate a signal that then travels through the spinal cord and nerves to reach a normally functioning muscle. Therefore, true weakness can result from injury or disease affecting the nervous system, muscles, or connections between them (neuromuscular junction).

Brain problems include strokes, injuries, tumors, and degenerative disorders (such as multiple sclerosis, which also can affect the spinal cord and nerves). Spinal cord disorders include injury, bleeding, and tumors. Spinal nerve roots can be affected by a ruptured intervertebral disk, and peripheral nerves by injury or polyneuropathy. The neuromuscular junction can be affected by myasthenia gravis, drugs such as botulinum toxin injections, and certain poisons such as organophosphates (used in nerve gas and many insecticides).

Muscle disorders causing weakness include muscular dystrophy and polymyositis. The muscle weakness that commonly occurs following immobilization (in a cast or from prolonged bed rest) and in old age is due to a reduction in muscle mass (sarcopenia) and results from lack of use. The remaining muscle mass functions normally, but there is not an adequate amount.

Weakness may be limited to one extremity or part of an extremity, as is typically the case when a single nerve, joint, or muscle is affected, or diffuse, as occurs in widespread neurologic or muscular diseases.

Pain: People with pain in the muscles, ligaments, bones, or joints tend to consciously and unconsciously limit motion. This often gives the impression of weakness even though the nervous system and muscles are able to generate movement.

Evaluation and Treatment

Doctors can often diagnose weakness based on the person's symptoms and the results of the physical examination. Doctors first try to determine whether the person can contract the muscles normally. If the person can contract the muscles normally but has trouble moving a joint, the doctor tries to move the joint for the person while the person relaxes (passive motion). If motion is painful, inflammation may be the problem. If passive motion causes little pain but is blocked, joint contracture (for example, due to scar tissue) may be the problem.

If passive motion is neither painful nor blocked, the person is giving full effort, and there is no sign of Parkinson's disease or other neurologic disorder causing difficulty initiating movement, then true muscle weakness is likely. The cause of true muscle weakness can often be determined by noting the person's symptoms, which muscles are affected, whether muscles have shrunk, and muscle tone and by testing the person's reflexes with a reflex hammer. For example, if weakness affects mainly the large muscles such as the hips, thighs, and shoulders, the cause may be a disorder producing widespread damage to

muscles. If weakness affects mainly the eye muscles (causing double vision), the cause may be a disorder of the neuromuscular junction. If weakness affects mainly the fingers, hands, and feet, particularly if there is loss of sensation, the cause may be a disorder that damages many nerves (polyneuropathy). The nerves to the fingers, hands, and feet are the body's longest and thus the most vulnerable peripheral nerves. If muscles have shrunk, the disorder causing the problem has been present for months or years. If the person's reflexes are decreased or slow, the cause may be nerve damage. If reflexes are increased or more rapid than expected, the cause may be spinal cord or brain damage. The doctor checks muscle tone by testing passive movement. Muscle tone may be decreased when weakness results from a peripheral nerve disorder. Muscle tone may be increased when weakness results from a spinal cord or brain disorder.

If the cause is still not clear, other tests can help. Disorders of the brain or spinal cord are diagnosed using neuroimaging tests such as CT or MRI. To differentiate between weakness caused by damage to the peripheral nerves, muscles, and neuromuscular junction, tests such as electromyography and nerve conduction velocity (see Diagnosis of Brain, Spinal Cord, and Nerve Disorders: Electromyography and Nerve Conduction Studies) usually help. Certain other disorders (for example, low blood levels of potassium or vitamin D) are diagnosed with blood tests.

For joints that are fixed, joint flexibility can be maximized by stretching exercises and physical therapy. If the joint's range of motion is severely restricted by scar tissue, surgery may be necessary. The only way to relieve weakness is to treat the disorder causing it.

Classifying Weakness

Underlying Problem	Example	Description
Muscle disease	Muscular dystrophies	A group of inherited muscle disorders that leads to muscle weakness of varying severity
	Infections or inflammatory disorders (acute viral myositis, polymyositis)	Muscles tender or painful and weak
Widespread muscle damage caused by use of a drug (drug-induced myopathy)	Myopathy due to corticosteroids, statins, lithium, alcohol, clofibrate, colchicine	Weakness usually begins at the hips and may spread to other muscles; pain may be absent
Low blood levels of potassium	Hypokalemic myopathy (caused by certain disorders or use of diuretics)	The person experiences periods of weakness throughout the body
Abnormal levels of thyroid hormone	High levels of thyroid hormone (hyperthyroidism) or low levels of thyroid hormone (hypothyroidism)	High or low levels of thyroid hormone produce weakness that is usually more pronounced in the shoulders and hips than in the hands and feet
Low levels of vitamin D	Osteomalacia	Pain in the back, with weakness in the legs; rarely pain throughout the body
Disease of the neuromuscular junction	Myasthenia gravis, curare toxicity, Eaton-Lambert syndrome, insecticide poisoning, botulism, diphtheria	Weakness or paralysis affecting all or many muscles; sometimes affects mainly eye muscles
Damage to a single nerve (mononeuropathy)	Diabetic neuropathy, local pressure	Weakness or paralysis of muscles and loss of sensation in the area served by the injured nerve
Damage to many nerves (polyneuropathy)	Diabetes, Guillain-Barré syndrome, folate deficiency, toxins, drugs	Weakness or paralysis of muscles and loss of sensation in the areas served by the affected nerves
Spinal nerve root damage	Ruptured disk in the spine of	Pain in the neck and

	the neck or lower back	weakness or numbness in an arm, low back pain shooting down the leg (sciatica), and leg weakness or numbness
Degeneration of nerve cell bodies in the spinal cord	Amyotrophic lateral sclerosis	Progressive loss of muscle bulk and strength, but no loss of sensation
Spinal cord damage	Trauma to the neck or back, spinal cord tumors, spinal stenosis, multiple sclerosis, transverse myelitis, vitamin B_{12} deficiency	Weakness or paralysis of the arms and legs below the level of injury, progressive loss of sensation below the level of injury, back pain; bowel, bladder, and sexual function are affected
Brain damage	Strokes, tumors, head trauma, multiple sclerosis, infections	Weakness or paralysis of muscles in the area served by the injured part of the brain, often with other symptoms of brain damage
Psychologic problems	Depression, imagined symptoms or hysteria (conversion reaction)	Complaint of whole-body weakness or paralysis with no evidence of nerve damage

JOINT STIFFNESS

Stiffness is the feeling that motion of a joint is limited or difficult. The feeling is not caused by weakness or reluctance to move the joint due to pain. Some people with stiffness are capable of moving the joint through its full range of motion. Joint stiffness usually occurs or is worse immediately after awakening or resting. Stiffness is common with arthritis. Morning stiffness commonly occurs with rheumatoid arthritis and other types of inflammatory arthritis in which stiffness typically occurs on arising and gradually lessens with activity only after an hour or two.

Doctors can sometimes diagnose the cause of stiffness by the person's symptoms and the results of a physical examination. The person is examined to make sure that the problem is not pain with motion or weakness. Because arthritis is often the cause, blood tests (for example, rheumatoid factor and antinuclear antibodies) and x-rays may be done.

Stiffness is relieved by treating the disorder causing it. Stretching, physical therapy, and taking a hot shower on arising may improve the ability to perform activities that require flexibility.

JOINT NOISES

Joint noises, such as creaks and clicks, are common in many people, but they can also occur with specific problems of the joints. For example, the base of the knee cap may creak when it is damaged by osteoarthritis, and the jaw may click in a person who has temporomandibular joint disorder. Doctors ask about the person's symptoms and perform an examination to determine whether a joint noise is a symptom of a certain disorder. Further evaluation and treatment are needed only if the evaluation suggests a significant joint problem. Joint noises themselves do not require treatment.

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